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Original Articles.

RECENT WORK ON THE CEREBELLUM AND ITS RELATIONS; WITH REMARKS ON THE CENTRAL CONNEXIONS, AND TROPHIC INFLUENCE OF THE FIFTH NERVE.[1]

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GENTLEMEN,—In assuming the office of President for the ensuing session, I desire, in the first place, to express my high appreciation of the distinguished honour, and to say that it will be my endeavour to promote to the utmost the best interests of the Society, and to maintain, with your help and co-operation, the high standard which the discussions and communications to the Society have attained.

It seems appropriate that on this occasion, the opening day of a new session, we should, as a branch of the universal brotherhood of science, pay some tribute to the memory of one recently passed away, whom we all, without exception, acknowledge to have been one of the greatest masters of our science in this or any age—the late Professor Charcot. It is altogether unnecessary that I should, before such an audience, attempt to recount the services which Professor Charcot has rendered to Neurological science. There is scarcely a subject

[1] Being the Presidential Address to the Neurological Society, January 25, 1894.

which he has not touched, and which he has not likewise adorned. His death, ripe in years and wisdom, and in the full enjoyment of his splendid reputation, has deprived the world of one whose place can never be filled, and our Society of one of the most illustrious of its small and select list of honorary members. Professor Charcot took much interest in our Society, in which he had so many personal friends, and on the last occasion when I saw him, shortly before his death, he promised to be present and take an active part at one of our meetings during the ensuing session. This, alas! to our grievous disappointment, was not to be. During the last year the Society has also lost in Dr. James Anderson, one of its office-bearers, esteemed alike for his qualities both of head and heart, and just as he was beginning to reap the fruits of the solid reputation he had established for careful and accurate clinical work; and in Dr. Walter Hadden, a neuro-pathologist of brilliant promise, an active participator in the discussions of the Society, and frequent contributor to its journal.

In inaugurating the work of a new session, it has been the custom of my predecessors to introduce some question of more or less present interest in Neurology in one or other of its various aspects. In accordance with this custom, which I consider well worthy of imitation, I have chosen as the chief subject of my discourse this evening one which, though old and worn, one would think, threadbare, is yet ever new and apparently as far as ever from being exhausted: to wit, the functional and anatomical relations of the cerebellum.

These have been practically reconstructed by the recent work of Luciani[1] and Marchi, and it is more particularly in reference to the views expounded by these enquirers, that I wish to engage your attention and consideration for a brief space. I have selected this topic from others that offered themselves, for the special reason that it has been my latest study, and has formed during the last two years a joint research with my colleague Dr. Turner. (*Proc. Roy. Soc.*, Dec. 14, 1893.)

[1] "Il Cervelleto," Firenze, 1891; *German Trans.*, Kleinhirn, 1893.

Luciani is the first who has, by skilfully contrived and well-executed experiments, succeeded in studying for long periods of time the symptoms consequent on complete or partial removal of the cerebellum in the higher animals; viz., dogs and monkeys; whereas, with the exception of fowls and pigeons, most previous experimenters had limited their observations to the more immediate effects of such lesions. But these, as the researches of recent years have more and more strongly emphasized, are of themselves inadequate data, and may even lead to erroneous conceptions as to the true functional relationships of any particular organ or nerve centre. The importance of Luciani's work cannot be over-estimated, and his analysis and synthesis are worthy of the most careful consideration.

The immediate effects of cerebellar lesion, as described by Luciani, and amply confirmed by our own experiments, do not differ materially from those which are familiar to everyone in the classical descriptions of Flourens. The stationary phenomena, that is, the phenomena of deficiency, are those which are particularly illustrated by abundant richness of experimental detail. By overwhelming proofs, if further proof were necessary, he has disposed finally of the supposed relation of the cerebellum to the sexual instinct. Indeed, as he himself says: "In the history of my experimental animals, there were so many love episodes, of such a wanton and grotesque character that one might really ask whether the loss of the cerebellum was not the cause, perhaps indirectly, of an abnormal degree of licentiousness; and these love relationships were not without their natural consequences. On the contrary, pregnancies, lyings-in, and parturitions followed each other so frequently that my laboratory appeared to have been converted into a maternity hospital."

There is no proof that the cerebellum has any share, or takes any part in psychical manifestations. The total removal of the cerebellum causes no discoverable impairment of any of the special senses, or of any of the forms of cutaneous or of muscular sensibility. In this respect our experiments are entirely in harmony with his.

Luciani has also furnished additional and conclusive evidence to show that the influence of the cerebellum is direct, and not crossed, and that each half of the cerebellum exerts its influence mainly, if not exclusively, on the corresponding side of the body.

His experiments, with which our own entirely agree, have summarily disposed of the theory of Nothnagel that to the middle lobe alone belong those functions which are commonly ascribed to the cerebellum, and that on lesion of this lobe, directly or indirectly, the symptoms of cerebellar disease exclusively depend. This view, which has always appeared to me a very strained reading of the facts of disease, in accordance with a preconceived notion founded on imperfect data, has been shown by Luciani's experiments, as well as our own, to be without foundation ; for the effects of removal of the middle lobe are not more pronounced, and are not more enduring than those which follow extirpation of the lateral lobe.

When the cerebellum has been totally removed from monkeys—an operation which we have found to be a most formidable one, and more frequently fatal than successful—the animals exhibit the most tumultuous disorders of equilibrium, so that station and locomotion are, for the time, altogether impossible. Gradually, however, these tumultuous disorders subside to such an extent that the animal is able to sit up, with the help of some support to which it clings, and ultimately regains such a degree of stability that it may dispense with extraneous aid, but is so tottery that it falls over on the slightest disturbance or excitement. Its gait is of a peculiar sprawling character ; at first, barely raising its abdominal surface from the ground, and planting its limbs wide apart so as to increase the basis of support ; these being in progression raised and set down in a brusque and characteristically ungraduated fashion, which character the movements of the limbs retain for an indefinite period. Notwithstanding this extraordinary unsteadiness and instability of equilibrium, the animals are, however, able to grasp tenaciously with both hands and feet, so much so that it is sometimes more easy to drag the chair to which

they may cling than loosen their grip; and they are able to climb a rope with agility hand-over-hand, in no way differing in this respect from perfectly normal animals. But the most noteworthy and persistent feature is the remarkable astasia or unsteadiness of the head, trunk, and limbs, which are either agitated by constant fine tremors, apart from obvious muscular exertion, or exhibit oscillations of the typical disseminated sclerosis type on volitional exertion; so that, for instance, an attempt to lay hold of a piece of fruit excites such wild oscillations of the hand and arm that the object aimed at is either knocked away, or, if seized, after many unsuccessful efforts, is with the utmost difficulty brought up and held to the mouth. This instability is a persistent feature, and, though lessening to some extent, never entirely disappears, and was clearly evident in one of Luciani's monkeys a whole year after the operation.

If only the lateral lobe, or one half of the cerebellum, is removed, the persistent symptoms, after the cessation of the primary disorders, namely, the sprawling, ungraduated action of the limbs, and the tremors or instability on volitional effort, are confined to the same side as the lesion.

If, on the other hand, the middle lobe is destroyed, or extensively injured, as by antero-posterior division, the symptoms are essentially of the same character as those which follow destruction of the whole organ, but they do not affect one side more than the other, and, according to our observations, are more pronounced in the head and trunk than in the limbs. They also appear to be less persistent, and, in the course of a few months, pass off to such an extent that, except on careful examination, it would be difficult to distinguish an animal so operated upon among its normal companions.

In the course of our investigations, Dr. Turner and I have succeeded in dividing the individual peduncles of the cerebellum with comparatively little injury to neighbouring structures at all calculated to complicate the symptomatology. When the primary disturbances, which I will allude to subsequently, have passed off, the persistent phenomena—whichever peduncle is cut—are essentially of

the same character as those following destruction of the lateral lobe, and are confined to the limbs on the side of lesion.

Such is a brief outline of the symptoms, temporary and permanent, following total or partial extirpation of the cerebellum and section of its peduncles. The question is, how are these to be explained in accordance with the anatomical connexions and relationships of the cerebellum? In answer to which I fear that, notwithstanding the valuable researches of Luciani, the opinion of Vulpian that "the problem of the functions of the cerebellum is still far from being definitely solved" is not inapplicable to the present state of our knowledge. Luciani's views are, briefly, as follows :—

The cerebellum, with its annexes, constitutes a more or less independent system. It is not an organ interpolated in the course of the cerebro-spinal tracts, but is an "end-organ," or, so to speak, an appendage to the cerebro-spinal axis, directly or indirectly related to certain peripherical sensory organs, and in direct efferent relationship with certain ganglia of the cerebro-spinal axis, and indirectly with the motor apparatus in general. It is functionally homogeneous, each part exercising the functions of the whole, but having special relation to the muscles on the corresponding side of the body. The symptoms following its destruction are, in the first place, *irritative* phenomena, opisthotonic or pleurosthotonic in character, according as the lesion is general or unilateral, together with rotation round the longitudinal axis, and squinting, or deviation, of the optic axis; secondly, *ataxic* phenomena, or phenomena of deficiency (Ausfallserscheinungen), which are more or less compensated by volitional effort on the part of the animal (functional compensation), or by such parts of the organ as have escaped destruction (organic compensation). The phenomena of deficiency, or cerebellar ataxy, are ascribed by him to three principal conditions, which he has a special fondness for reiterating; to wit, *asthenia*, *atonia*, and *astasia*, due to the loss of a sthenic tonic, and static influence, which, according to him, the cerebellum normally exerts on the apparatus of

movement, and which is the essence of its function. In addition to this, he ascribes to the cerebellum a trophic function, direct on the efferent tracts which spring from it, and which, therefore, degenerate when the cerebellum is destroyed, and indirect on the nutrition of the body generally.

I should like, with your permission, to examine some of these views a little more in detail.

Touching the so-called irritative nature of the primary disturbances (Reizerscheinungen), which Luciani ascribes to inflammatory action set up by the lesion ("Kleinhirn," p. 151), I am persuaded that this is erroneous, and would prefer with Goltz to term them dynamic, or Hemmungserscheinungen, without attempting to offer a more exact explanation of the mode in which they are brought about. They certainly occur under conditions which entirely exclude inflammatory action, and under methods which, when applied to the cerebral cortex, cause paralytic and not irritative symptoms; and, moreover, there is reason to believe that the phenomena are entirely reversed when the conditions are such as to excite true vital irritation or inflammatory action. The so-called irritative phenomena occur, according to Luciani, more especially when the peduncles are injured, and this is, on the whole true; for, according to our experiments, rotation round the longitudinal axis certainly occurs more constantly when the peduncles are divided than when the lateral lobes are removed. But this is not an absolute rule, and one of the most marked instances of rotation round the vertebral axis which we observed was an animal in which only the cortex of the lateral lobe had been partially cauterized, and under conditions likely to excite irritation of neighbouring parts. In this case the rotation was exactly in the opposite direction to that which usually occurred. The direction of the rotation appears to be the subject of some confusion and difference of opinion. Magendie, as is well-known—and his observations have been confirmed by Hitzig and others—described the rotation after section of the middle peduncle as taking place towards the side of lesion; so that, for instance, if the left were cut

the animal would roll to this side with incredible force and rapidity. Luciani, however, describes the rotation as uniformly occurring from the injured to the sound side, so that, *e.g.*, if the right side of the cerebellum is removed, the rotation takes place from right to left.

Our experiments on the cerebellar peduncles, effected in a precise manner by simple section, are absolutely in harmony with the results obtained by Magendie and Hitzig. We have found that, without exception, the animals roll towards the side of lesion.

The apparent discrepancy between our results and those of Luciani has been so uniform that we cannot but think that it is more in the manner of description than in the actual facts themselves. The facts, according to our observations, are that the animals, when placed on the ground, roll towards the side of lesion, as if carried by some irresistible force. And yet the impulse which causes the animal to roll towards the side of lesion, may be correctly described as a rotation round the vertebral axis from the injured to the sound side. Thus, if the left peduncle is divided, the animal will roll towards the left, but the impulse which causes this rolling movement would, if the animal were viewed in front, appear as a rotation round the vertebral axis from left to right. It might, therefore, be equally correct to say that the *rotation* is from the injured to the sound side, and that the *rolling* is towards the side of lesion. That this is the real explanation of the apparent discrepancy, I gather from a passage in which Luciani gives a detailed account of what he terms rotation from left to right. He says ("Kleinhirn," p. 139): "When the animal [a dog] was laid on its left side the torsion of the vertebral column was such as to bring the dorsal surface downwards, and the feet in the air. From this position, and with movements of all four limbs, it turned on its right side with its back to the right, and then on to its left side." It appears to me evident that if these movements were repeated several times in succession the result would be that of an animal rolling towards the left side. If this is not the true solution of the seeming discrepancy as to the direction of the rotation, as described by

Magendie and Luciani, we should say that our results are in agreement with those of Magendie, and that the direction in which animals roll after removal of the cerebellar hemisphere, or section of its peduncles, is towards the side of lesion.

The deviations of the optic axes, described by Magendie in connection with the rotatory phenomena, we have found in monkeys to be far from uniform, and we have not been able to establish any constant relation between lesion of any particular portion of the cerebellum, and any particular distortion or deviation of the optic axes. Nor have we found those indications of tonic contracture of the limbs in monkeys on the side of lesion such as Luciani appears to have uniformly observed and recorded as signs of irritation.

A special and characteristic attitude is assumed by animals after section of one or other peduncle, namely, curvation of the vertebral column, with the concavity towards the side of lesion, adduction and flexion of the limbs on the same side, and abduction and extension of the limbs on the opposite. The chin, as a rule, deviates towards the sound side, that is, in the direction of the rotation which causes the animal to roll towards the side of lesion. But the flexion of the limbs on the side of lesion is not a rigid flexion or contracture, and offers no resistance to passive movements or change of position.

Let us now examine what Luciani considers to be the essential factors of cerebellar ataxy, namely, asthenia, atonia and astasia.

First, as to asthenia, by which is meant a defect of energy, due to the loss of some influence supposed to be exerted by the cerebellum, which heightens or reinforces the energy of muscular contraction. Of the existence of any such influence, such as has been contended for by Luys, Weir-Mitchell and others, or that the phenomena of cerebellar ataxy can in any way be ascribed to such a condition, I have discovered no satisfactory evidence, either from a study of Luciani's experiments, or in the facts of our own, or in the phenomena of cerebellar disease in man. On the

contrary, clinical observation has abundantly shown that in cases of atrophy or disease of the cerebellum, of such a nature, and of such extent, as to render station and locomotion altogether impossible, the patients may in the recumbent position perform all movements of the limbs with energy and vigour. Of one of his patients, who was suffering from atrophy of the cerebellum, and who could not walk except in the most bizarre fashion, pushing a chair in front of her and often falling down, Vulpian remarks: "Yet, and this is very remarkable, she retained such muscular vigour that when one held out a hand to save her from falling she gripped so firmly as to cause pain."

When a monkey, within a few days after complete removal of its cerebellum, and so unstable as to be unable to sit up without support, or progress except in a sprawling fashion with its abdomen in contact with the ground, is, notwithstanding, able to climb a rope or lattice work hand-over-hand with apparently the same agility as a normal monkey, and grips so firmly with hands and feet as to require considerable force to detach it, I do not think the term "asthenia," or defect of muscular energy, is at all applicable to such an animal. And when a monkey, within an hour of removal of the left lobe of the cerebellum, and still unable to sit up, is able to grip so firmly with its left hand as to bear the whole weight of its body on it, I do not think that its left limbs can at all properly be described as being affected by asthenia. Yet these and similar facts might be multiplied from our observations.

In no case in which either the lateral lobe was removed, or a peduncle cut, could we detect any appreciable difference in the grip of the two sides. We found it, however, difficult to make exact dynamometric observations, as the animals were not always in the humour to make that trial of their strength which alone would permit of accurate comparison, and without which tests of this kind are altogether fallacious. There is, however, no doubt that an animal after removal of one lateral lobe prefers to use the limbs on the sound side, but this is obviously more in consequence of the instability of the limbs than from any real want of strength, and such,

in my opinion, is the real explanation of the apparent want of strength in the limbs on the side of lesion which Luciani has so frequently noted. For the most part, he speaks of a semblance of weakness only, as evidenced by the doubling up of the limbs and tendency to fall on the injured side. But when the same monkey which exhibits this defect is able to climb well, and a dog to swim vigorously, as he admits, the symptom cannot be explained by loss of muscular energy.

One of the proofs relied on by him as indicating relative weakness of the limbs on the side of lesion is of so remarkable a character as to deserve special mention. Thus, he says that dogs deprived of one-half of the cerebellum, when swimming, tend, almost invariably, to the opposite side. Thus, if the right half of the cerebellum has been removed, the animal swims towards the left, in consequence, he says, "of the greater energy of movement of the limbs on the left side." ("Kleinhirn," p. 171.) Any swimmer, however, would say that in order to swim to the left he would require to make more vigorous efforts with his right limbs, and the very fact mentioned by Luciani would appear to prove exactly the opposite of what he says. In further corroboration of this may be mentioned another observation of his, viz., a dog in which the right side of the cerebellum had been removed, together with the motor centres (sigmoid gyrus) of the left hemisphere, thereby causing paresis of the right limbs. This dog in swimming, swam towards the right side, because, as he says, and truly, "the left limbs struck the water more vigorously than the right." (*Ibid.*, p. 133.)

With these remarks I would dismiss Luciani's theory of asthenia as an element in the causation of so-called cerebellar ataxy, as being unsupported by valid evidence. That animals after complete removal of the cerebellum may, as Weir-Mitchell says, show signs of exhaustion after sustained muscular effort sooner than normal animals, is, I believe, more easily explained on my own hypothesis that the efforts at functional compensation, effected, as Luciani has verified, by the cerebral motor centres, are a considerable strain on

the animal's power, and, in consequence, lead to exhaustion much sooner than under normal conditions.

Next, as to the evidence of atonia, or loss of tone in the muscles. The usual test of the tone of muscles is the readiness, or otherwise, with which they respond to mechanical tension, such as is constantly exemplified in the usual method of eliciting the patellar-tendon reaction. Luciani has, however, apparently not investigated the condition of the tendon reflexes in his experimental animals. As a proof of the loss of tone, he speaks of the muscles of the limbs on the side of lesion as appearing to him more flaccid, and of the leg occasionally giving way, as if the tone suddenly ceased, &c., all of which appear to me more matters of constructive speculation than actual realities.

That the tone of the muscles, so far at least as indicated by the tendon reactions, is not diminished by cerebellar extirpation, complete or unilateral, or after section of its peduncles, has been amply proved by our own experiments, as well as by those of Dr. Risien Russell. (*Proc. Roy. Society*, vol. liii., p. 430.) We have not, however, observed the marked exaggeration of the knee-jerk on the side of lesion, which Dr. Russell appears to have found in dogs. As a rule, little or no difference could be made out after extirpation of the lateral lobe, or section of the peduncles on one side, immediately after the operation. If anything, the difference was in favour of the reaction on the side of lesion, but it showed a distinct tendency to increase the longer the animals survived; and when the whole cerebellum was extirpated, the knee-jerks were, after some months, decidedly increased, as compared with the normal, but there was no rigidity of the limbs.

These facts lead me to diverge for a moment to comment on the views propounded by Drs. Hughlings Jackson and Bastian as to the mechanism of the development of rigidity in limbs paralysed by transverse lesions of the spinal cord. Dr. Bastian has adduced weighty evidence (*Med. Chir. Trans.*, vol. lxxiii., p. 151), and he has been amply supported by Bowlby (*Ibid.*, p. 313), and others, to show that in total transverse lesions of the spinal cord the tendon reflexes are

abolished and never return, and that this condition cannot be satisfactorily explained on the hypothesis of a coincident lesion, or mere dynamic disturbance of the centres situated below the lesion. Though I think the point is one which may well bear further investigation, I am prepared, for the purposes of argument, to admit Dr. Bastian's law as being universally applicable. But his further proposition to the effect that the development of rigidity in limbs paralysed from cerebral disease, or partial lesion of the cord, is due to the "unrestrained," or, as Dr. Jackson would say, the "unantagonized" influence of the cerebellum passing downwards, perhaps through the grey matter of the cord, I cannot allow to pass without question.

First, I can find no experimental proof that the cerebellum exercises any such influence as that contended for by Dr. Bastian. It is no doubt true that in some cases of disease in the posterior fossa, involving the cerebellum, the tendon reflexes are abolished. I am not acquainted with, and am myself unable to offer any satisfactory explanation of this fact, but, certainly, it is not the rule that the knee-jerks are abolished in cerebellar disease. And when we find that in monkeys, after complete removal of the cerebellum, the knee-jerks instead of being lost become actually increased, it is clear that the mechanism for the increase of the knee-jerks still exists. That this is independent, both of the cerebrum and cerebellum, is further evident from the fact that in monkeys the knee-jerks may become increased, and late rigidity ensue, when the spinal cord has been totally divided transversely. In illustration of this point, I will here briefly mention the facts of two experiments made by Dr. Turner and myself.

In the first case, the spinal cord was divided transversely and completely at the level of the eighth dorsal root in March, 1891. Immediately after the operation the knee-jerks were capable of being elicited as before the operation. The animal exhibited the usual paraplegic symptoms, both as regards total paralysis of motion and sensation, and continued so until its death on July 24, that is, fully four months. During this period there

gradually developed well-marked increase of the knee-jerks, as well as of the superficial reflexes, together with considerable rigidity of the limbs. The animal was exhibited before its death to Drs. Hughlings Jackson and Bastian, who both satisfied themselves as to the condition here described.

In a second case, the cord was completely divided at the level of the sixth dorsal nerve on July 21, 1891. Both knee-jerks were readily elicited immediately after the operation, if anything, more markedly than before. The animal lived till September 29; that is, two months. This animal, however, exhibited some noteworthy differences as compared with the former. Instead of a gradual increase of the knee-jerks and development of late rigidity, no reaction could be obtained on August 4, that is, twelve days after the operation. The knee-jerks proper remained in abeyance till the 20th, that is, for sixteen days. During this time the muscles of the limbs had undergone considerable wasting, and the faradic contractility of the quadriceps extensor became decidedly diminished as compared with the other muscles, and more so on the left than on the right. After the 20th the right knee-jerk re-appeared and continued to increase. The left remained in abeyance until September 10 at least, after which date the knee-jerks were not examined, as the animal died during the holiday season.

The first of these two cases shows conclusively that in the cord itself there exist the conditions for the presence and increase of the knee-jerks, and the subsequent development of rigidity or contracture of the limbs. And the second case shows also, in a highly instructive manner, that in the cord itself conditions may occur which, either temporarily, or it may be permanently, prevent the manifestation of these phenomena. In both these cases, the influence of the cerebrum and cerebellum was, from the first, entirely excluded. But it will probably be said that though this may be true of monkeys, it is not applicable to man. That the symptomatology of total transverse lesions in man and the lower animals is widely different, I readily admit. Indeed, we have every reason for believing that the independence of the

spinal centres is much greater in the lower animals than in man. We all know the extraordinary adaptiveness and apparent intelligence of the spinal cord of a frog; and the experiments of Goltz on dogs have shown that, even in animals so high in the scale as dogs, the hind limbs, after total transverse section of the spinal cord, remain capable of a number of the most complex, reflex or responsive actions. In answer to some enquiries I made of him in regard to his animals, Professor Goltz wrote to me: "Several of my dogs learnt to stand on their hind legs, and even to walk a few steps. While so standing, the hind legs became stiff in consequence of a general contracture of the muscles of a reflex character, conditioned by the position of the animal." Such things are, of course, impossible in monkeys and, *a fortiori*, in man.

The point, however, which I desire to emphasize is, that the increase of the knee-jerks, and development of rigidity, can occur in monkeys altogether independently of the cerebellum, owing to conditions in the cord itself, in which also the opposite conditions may occur, either temporarily or permanently. The difference between the symptomatology of total transverse lesions of the cord in man and the lower animals is, in my opinion, due to the greater independent vitality of the spinal centres in the latter as compared with the former. In these, total separation from the higher centres leads to such impaired vitality, that they are no longer capable of manifesting that increased excitability, which is the primary cause of the phenomena in question.

Let us now return to the third cardinal factor of cerebellar deficiency; namely, the condition which Luciani terms "astasia." This term very aptly describes the primary instability, both of station and locomotion, as well as the subsequent, and more persistent, tremor and unsteadiness, not only of the body as a whole, but of the limbs, which is so noteworthy a feature after cerebellar extirpation in monkeys and dogs. The reeling gait and the various degrees of what most nearly represents alcoholic intoxication, are familiar and generally recognised symptoms of cerebellar

disease in man, but the tremor and unsteadiness of the limbs, though occasionally noted, and more particularly in cases of cerebellar atrophy, are not so common, and yet they are sometimes present, as I have myself had occasion to observe. When they occur more particularly on one side, they ought to furnish a reliable guide to the determination of the position of the lesion, inasmuch as we have seen that they occur specially on the side of lesion. It would appear, therefore, that the instability of cerebellar defect is not limited to the muscles of the trunk, but affects the muscles, in general, and manifests itself not only in the unsteadiness on volitional effort, but also in the ungraduated or dismetric (Luciani) manner in which the limbs are raised and planted in progression, in spite of the most complete compensation which the voluntary motor centres are capable of effecting. But the term "astasia" is, after all, only a description, better perhaps than any other that has been applied to the phenomena of cerebellar deficiency, and not an explanation; and the problem still remains, how is the astasia conditioned? To explain this is to my mind to explain the true mechanism of the cerebellum.

Luciani sees in the tremors and oscillations, &c., a defective summation of the individual impulses on which the muscular contraction depends, and he looks on the abruptness of the movements of the limbs as due to a sudden flaccidity (Erschlaffung) of the antagonists while the flexors or extensors are acting respectively; or, it may be, owing to collateral muscles intervening which are not generally concerned in the action in question. And there is much to be said in favour of a hypothesis of this kind. And yet I question whether this is any improvement on the hypothesis of Herbert Spencer, that the cerebellum is the organ of "doubly compound co-ordination in space," while the cerebrum is the organ of "doubly compound co-ordination in time," so often and so ably expounded and applied by Dr. Hughlings Jackson. "According to this hypothesis," as James Ross has lucidly stated it, "all the muscles of the body are innervated both by the cerebrum and cerebellum,

but in an inverse order. The cerebellum regulates the muscular contractions necessary for the maintenance of our attitudes in space, while the cerebrum regulates the contractions necessary to effect all the change of attitude which are made in response to the successive impressions occurring in time. Now, so long as a particular attitude is maintained in opposition to gravity and other forces, the contractions of the various groups of muscles concerned must be continuous and in equilibrium with each other, while each change of attitude necessitates the overthrow of this equilibrium, involving the preponderance of the contractions of some groups of muscles over those of others, so that the change of attitude involves alternate muscular contractions and relaxations. Speaking broadly then, the cerebellum regulates continuous or tonic muscular contractions. It will be seen, therefore, that every compound muscular adjustment necessitates the co-operation of both these organs. No change of attitude can be effected by the cerebrum, except in so far as a certain attitude was previously maintained by the cerebellum, and no steady movement can be produced by the alternate contractions of some groups of muscles except in so far as other groups of muscles are maintained in a state of continuous contraction; hence it may be inferred that all the movements of the body are co-ordinated both in the cerebellum and cerebrum." (Ross, "Diseases of the Nervous System," vol. i., p. 57.) The cessation of the tonic or continuous influence of the cerebellum should, therefore, exhibit itself in an unsteady, ungraduated character of all the motor adjustments, whether of the body as a whole, or of its individual parts, and would, theoretically, produce a state of affairs which agrees well with the actual effects of cerebellar extirpation in animals.

Even, however, if we assume that this is the true formula for the influence of the cerebellum, it still remains to be determined how its activity is called into play and brought to bear on the muscles, either in association with the cerebrum, or independently. That it is a more or less independent responsive mechanism is clearly shown by the fact that the stability of motor adjustments is still

capable of being maintained after the cerebrum has been removed. That the cerebellum stands in complex afferent and efferent relations with the cerebro-spinal system is undoubted, but we, as yet, know little as to the true functions of the respective centripetal tracts, and less, perhaps, with regard to the centres and tracts through which it acts centrifugally.

The superior peduncles proper appear to be efferent in function; that is to say, after cerebellar extirpation they degenerate *from* the cerebellum up to the opposite red nucleus and thence to the optic thalamus. On the dorsal aspect of the brachium conjunctivum, however, there is a tract which degenerates *towards* the cerebellum, a tract which the observations of Loewenthal,[1] Mott,[2] Tooth,[3] and our own, prove to be the termination of Gowers' antero-lateral spinal tract. As yet, however, there has been no exact determination of the functions of the red nucleus, or of Gowers' antero-lateral tract.

The middle cerebellar peduncle appears also to be efferent from the lateral lobe of the cerebellum to the opposite nucleus pontis. It is, no doubt, through the intermediation of the cells of the nucleus pontis that the cerebellar hemisphere of the one side is brought in relation with the cerebral hemisphere of the opposite. But here again we are in ignorance as to the true signification of those tracts, which are described as connecting the frontal and temporo-occipital regions of the cortex with the cell groups in question.

The inferior cerebellar peduncle contains both efferent and afferent fibres, *efferent* to the opposite inferior olive, by way of the external arcuate fibres; and *afferent*, viz., the direct cerebellar tract from Clarke's vesicular column, largely also of fibres from the posterior median and postero-external columns of the spinal cord by way of the clavate and cuneate nuclei, and probably also of the vestibular branch of the eighth, and, perhaps, also some of the other cranial nerves. But as to the exact functions of these different afferent

[1] *Rev. Med. d. l. Suisse Romande*, 1885.
[2] BRAIN, vol. xv., 1892.
[3] *Ibid.*

tracts to the cerebellum, we know very little. No discoverable loss of sensation ensues when they are divided. In particular, destruction of the clavate and cuneate nuclei, though leading to temporary disturbances of equilibrium, causes no discoverable impairment of any of the forms of cutaneous or general sensibility. And as the great bulk of the fibres which spring from the clavate and cuneate nuclei decussate in the interolivary layer, and ascend as the mesial fillet, which degenerates completely when they are destroyed, it is clear that this is not the path of transmission of sensations proper to the cerebrum. A tract which forms the internal division of the restiform body, and termed by Edinger "the direct sensory cerebellar tract," has been supposed to be the path by which the fifth, eighth, and probably also the vagus and glosso-pharyngeal nerves form connections with the middle lobe of the cerebellum. We have found, however, after extirpation of the middle lobe, that the tract in question degenerates downwards, or in an efferent direction, towards Deiters' nucleus. This is the only tract which appears to degenerate centrifugally from the middle lobe, a fact which shows that this lobe has no direct connection with the efferent tracts, which emerge in the cerebellar peduncles. Beyond those already mentioned, there is no proof of the existence of any other efferent tract from the cerebellum to the spinal cord. Marchi,[1] however, has described a tract which degenerates downwards in the antero-lateral region of the cord after extirpation of the cerebellum, which he derives specially from the middle lobe by way of the posterior longitudinal bundles and fillet. The tract described by Marchi occupies the periphery of the antero-lateral region, with an offshoot posteriorly projecting into the lateral column. Our observations, however, lead us to believe that Marchi's views as to the origin of this tract are erroneous; for in a case of total extirpation of the cerebellum, which lived several months, we found no evidence of any degeneration in the position assigned by Marchi to his tract. And yet in one case of removal of the lateral lobe we found a degeneration in the periphery of the anterior

[1] *Sul. Orig. e. Decor. dei Ped. Cerebell.*, 1891.

column corresponding in position with the anterior division of Marchi's tract ; and in another we found a well-marked descending degeneration in the lateral column in the region of the posterior division of the tract described by him. A further careful examination, however, of these cases, indicated that the degeneration in the anterior column was due to lesion of Deiters' nucleus, which would thus be an internode between the middle lobe and the anterior marginal column of the spinal cord, and that the degeneration in the lateral tract stood in relation to lesion of the lateral fillet, which forms a tract connecting the posterior corpus quadrigeminum with the lateral column of the cord. But while so much obscurity still prevails as to the functional significance of the various afferent and efferent tracts which connect the cerebellum with the rest of the cerebro-spinal system, we are not yet in a position to go much beyond mere speculation as to the mechanism of cerebellar co-ordination. In this some of us have indulged with greater or less plausibility, but I do not propose on this occasion to enter on the field of speculation, as I think we may more profitably wait for the dry clear light of further exact research.

If I may trespass on your patience a little longer, I should like to say a few words on a question which, though not directly related to the main subject of my discourse, has yet formed the subject of some inquiry in the course of our other experiments on the connexions of the cerebellum. Thus, on section of the inferior peduncle, it not unfrequently happened that the immediately subjacent, so-called, "ascending root" of the fifth nerve was wholly or partially severed. The result of lesion of this root was more or less complete anæsthesia of the cornea, and of the region of distribution of the sensory branches of the fifth nerve of the head and face. And by other experiments, not necessary to be detailed here, it was proved that this so-called ascending root is the true sensory root of the fifth nerve, and is, in reality, a descending root springing from the Gasserian ganglion, and passing through the substantia gelatinosa Rolandi towards the cells of the posterior cornu as far, at least, as the origin of the second cervical nerve.

On dividing the superior peduncle, one usually severs also the fibres of the so-called "descending root," which courses underneath this peduncle towards the crescentic layer of cells which surrounds the grey matter of the aqueduct of Sylvius. After such section, as well as after section of the motor root within the pons, but not after section of the sensory root, there followed atrophy of the fibres of this root, and of the crescentic layer of cells above-mentioned. The atrophy, however, was not a degenerative, but a simple atrophy, similar to that which occurs, as has been shown by Bergmann,[1] Darkschewitsch,[2] &c., in the central end of a motor root after division of the trunk. This is the root which has been supposed by some to join the sensory, and by others to join the motor division of the fifth: while Merkel has described it as being the trophic root of the trigeminus. This also is the root which Mendel[3] has described as being atrophied in a case of old-standing facial hemiatrophy. A recent experiment of ours points to the descending root as being the motor root *par excellence* of the fifth nerve. No loss of sensation was caused in the cornea or elsewhere by its division, nor did any trophic disturbance occur in the eyeball during the many weeks which the animal survived. On the other hand, atrophic paralysis ensued in the muscles of mastication.

A point which we kept specially before us in our experiments on the fifth nerve, was the so-called trophic influence of this nerve on the eyeball, a question which is still the subject of dispute, and which has come more recently again into prominence in connection with surgical operations on the Gasserian ganglion, with a view to the relief of inveterate trigeminal neuralgia.

The question is, as to the exact causation of the ulceration of the cornea and subsequent destructive inflammation of the eyeball, which have been so frequently observed both in man and animals after lesion or section of the fifth nerve. Mere anæsthesia, with the consequent loss of the protective

[1] *Obersteiner's Arbeiten*, 1892.
[2] *Neur. Centralblatt*, 1893.
[3] *Neurol. Centralbl.*, 1888.

influence conferred by the adaptive reactions of the eyelids, does not seem capable of explaining the facts, for the eye may be insensible from cerebral lesion, or even from lesion of the fifth nerve itself, and yet show no sign of corneal ulceration or destructive inflammation, though the anæsthetic eye has not been more protected from external influences than the other. An interesting case of this kind has been recorded by Hutchinson (*Ophthal. Hosp. Reports*, vol. iv., p. 191), and many others might be alluded to. And the acute inflammation has been seen in cases of lesion of the fifth nerve, not sufficient to cause complete anæsthesia ; facts which have led Meissner and others to postulate the existence of a separate set of fibres exercising a trophic influence on the eyeball, apart from those which confer sensibility on it. Nor can the inflammation be satisfactorily accounted for by mere vascular paralysis, for the inflammatory disorders first show themselves in the cornea, the extra-vascular part of the eyeball ; and Sinitzin states, though in this he is opposed by Eckhard,[1] that section of the cervical sympathetic, and consequent vaso-motor paralysis, actually prevents the inflammatory consequences otherwise following section of the fifth. Snellen found that when the eyeball was completely protected no inflammation occurred ; and he, therefore, attributed the whole of the phenomena to external influences. But these conclusions have been contradicted by others (Decker, Laborde, Büttner, &c.) who have observed speedy destructive inflammation follow section of the nerve in spite of protective precautions ; and also cases in which the eye did not become inflamed, though no special precautions were taken to protect it. There remains, therefore, some factor requiring elucidation which is of considerable importance from a practical point of view.

Magendie first pointed out, and in this he was confirmed by Longet and others, that the effects of section of the fifth nerve differ according as the lesion was made behind or in front of the Gasserian ganglion. Thus, whereas, section between the ganglion and the pons did not, or rarely, cause

[1] *Beiträge* bd. x., " Phys. d. Nerv. Trigem."

inflammation of the eye, section anterior to it was usually followed by destructive panophthalmitis. The latest experimenter in this field, Gaule (*Centralblatt für Physiologie*, 1892, p. 419) confirms the statements of Magendie, and holds that only lesion of the Gasserian ganglion itself, or section of the ophthalmic division, is effectual in inducing trophic disorders. These, he holds, occur immediately on section of the nerve, and altogether irrespective of external influences. These trophic disorders consist, according to him, in necrosis of certain portions, with cell proliferations in other portions of the cornea, as well as changes in the corneal corpuscles, Descemet's membrane and aqueous humour, which necrotic and correlated changes, in consequence of external influences, such as drying, mechanical injuries, septic infection, &c., advance to the well-known destructive neuro-paralytic ophthalmia.

Our experiments have included section of the so-called ascending root in its course above and through the tubercle of Rolando; section of the trunk of the nerve between the ganglion and the pons; and section of the ophthalmic division in front of the Gasserian ganglion. No special precautions were taken to protect the eye on the side of lesion, which was simply left under the same conditions as the normal one.

Of twelve experiments in which the nerve was divided behind the ganglion, the duration of life was from two days to four months. With the exception of two, which deserve separate mention, none of these cases exhibited any progressive ulceration of the cornea or destructive panophthalmitis. Of those that exhibited any alteration, there was, at most, some degree of conjunctivitis, and haziness of the cornea, as if from drying of the surface. In the majority the cornea remained quite clear and transparent. In one of the exceptional cases above alluded to, in which the eye remained in a perfectly normal condition for eight days after the operation, a drop of collodion accidentally entered the anæsthetic eye. This occasioned intense conjunctivitis, œdema of the eyelids, and a milky white opacity, which overspread the whole of the cornea. Mr. Gunn, who examined this case

with us, attributed the opacity of the cornea to the shedding of the superficial epithelium, similar to that produced in man by an irritant, such as lime. The opacity, however, disappeared in the course of a fortnight, and the cornea regained its transparency with the exception of a minute ulcer surrounded by slight opacity, which remained till the death of the animal, six weeks after the operation.

These cases show, therefore, that there is no necessary tendency to progressive inflammation of the eyeball after division of the fifth nerve, behind the Gasserian ganglion, and that such changes as occur do not differ in their process of repair from those that occur in a perfectly normal eye. In one case, after division of the trunk of the nerve, the cornea on the second day exhibited a depressed ulcer and some degree of opacity, which, however, cleared somewhat at the end of a week, but again set in accompanied by a purulent discharge, which resulted in complete disorganization of the eyeball in the course of six weeks. In this case, however, there were indications that the wound was not strictly aseptic, and the temperature remained throughout higher than normal. The probability is, therefore, that the progressive destructive inflammation of the eye was due to septic irritation of the trunk of the nerve.

The most instructive and interesting of our experiments are those relating to division of the fifth, between the Gasserian ganglion and the eyeball. This is the lesion which most observers have found to be specially calculated to excite inflammatory changes in the eye, apparently independent of external influences. We have divided the ophthalmic division in front of the Gasserian ganglion in five cases. In three of these animals, which lived respectively two days, fourteen days, and six weeks, the cornea remained perfectly clear and transparent during the whole time of the animals' survival. Microscopical examination, which was carried out by Dr. Bulloch, showed that the epithelium, corneal corpuscles, and substance of the cornea presented a perfectly normal appearance. In the one which lived six weeks, the anæsthetic cornea did not take on the chloride of gold stain like the normal, but as regards other re-agents

there was no change. Occasionally the cornea, when examined by a magnifying glass, was seen to exhibit minute, pin-pointed depressions, such as described by Gaule and Eckhard, as if from partial shedding of the superficial epithelium, but similar depressions not unfrequently occurred also in the sound eye, and they did not appear to have any pathological significance.

In a fourth case the cornea, which had remained perfectly clear and transparent until the fourth day, was irritated by a minute speck of nitrate of silver, and exactly the same condition was established also in the normal eye. This gave rise in each eye to a superficial ulcer with mucopurulent conjunctivitis, but there was no progressive ulceration of the cornea in either case, and the process of repair occurred in the anæsthetic eye as well as in the normal one. In a fifth case, however, the cornea, which had remained clear for two days after the operation, exhibited at the end of that time a central depression with opacity, which progressed until it overspread the whole surface. In this case, however, there were indications of septic irritation in the wound with febrile disturbance.

It results, therefore, from these experiments, that the separation of the eye from the Gasserian ganglion by division of the ophthalmic branch of the fifth, does not necessarily induce any trophic disturbance in the eyeball, or interfere with the processes of healthy nutrition and repair. The only strictly trophic influence of the Gasserian ganglion is, I conceive, on the cellulipetal processes of the Gasserian ganglion cells and their annexes, which probably undergo atrophy, though we have not as yet been able to demonstrate this microscopically. There seems little doubt that the so-called trophic disturbances, which so commonly occur after division of the ophthalmic branch, are due to inflammatory irritation of the nerve, and are of the same character as those which occur in connection with similar irritation of sensory nerves generally. By this irritation, changes are indirectly excited in the corneal tissues, which, under the influence of external agencies and septic infection, result in general panophthalmitis. If, however, proper measures are

taken to avoid setting up inflammatory irritation, there is no reason, so far as I can see, why extirpation of the Gasserian ganglion, or section of the ophthalmic division, should excite inflammation of the eye any more than that section of any sensory nerve should be followed by so-called trophic disorders in the region of its distribution, which we know practically is neither a necessary nor, indeed, a common occurrence.

ON HÆMATOMA OF THE DURA MATER ASSOCIATED WITH SCURVY IN CHILDREN.

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THE manifestations of scurvy in children present some differences from those seen in the case of adults. The most important of these are the absence of sponginess of the gums, and the presence of extensive sub-periosteal hæmorrhages in connection with the long bones. The explanation of these variations is to be found, I believe, in the fact that rickets is almost always present, and leads to considerable alterations in the vascular supply of those parts in which scorbutic changes are most marked. In rachitic infants the growth of the teeth is at a standstill, due probably to a diminished blood supply, and sponginess of the gums may not appear during an attack of scurvy. As regards the bones, in rickets there is a greatly increased vascular supply, as shown by the marked periosteal activity, and the thickening around the epiphyses. If scurvy supervene, the hæmorrhagic tendency will naturally show itself in those places where the vascular supply is greatest, and consequently periosteal and epiphysial hæmorrhage is a prominent sign. For a similar reason the active changes present in the skull in rickets seem likely to lead to hæmorrhage in that region if scurvy is present. The occurrence of hæmorrhage under the pericranium has been noted by several observers. With the exception of a doubtful case of Möller's, quoted by Dr. Barlow, I have not met with any recorded case of infantile scurvy in which intracranial hæmorrhage was present. My own experience is limited to the following two cases which

were admitted into Paddington Green Children's Hospital. I have to express my indebtedness to Dr. Leslie Ogilvie for permission to publish these notes, and to Dr. Leonard Guthrie for his reports of the *post-mortem* examinations.

Case 1.—*Female child, aged two years: anæmia; fracture of humerus and femur; periosteal effusion; bruise-like extravasations; death; autopsy; hæmorrhage into the brain and its membranes.*

This patient was admitted to hospital on March 21. The child had been healthy when born, and was fed on breast milk entirely for four months. Since that time her food had consisted almost entirely of Nestlé's milk. There had been no snuffles or rash observed. Four months previously she had been put out to nurse. The nurse stated that in the beginning of February the child seemed to have great pain in the right thigh, which was swollen, and she took her to a hospital where disease of the hip joint was diagnosed. Two days before admission the mother got the child back, and noticed a swelling along the right thigh, and also along the left upper arm. There was also a bruise-like discolouration over the right side of the forehead.

The patient was an only child. The father and mother were healthy, and there had been no miscarriage.

On admission the child was found to be fairly well nourished, and had a pale waxy complexion. There were some dirty looking sores along the upper lip, and alæ nasi, and also on the buttocks. A large bruise-like mark was present on the right frontal eminence, greenish in colour. The signs of rickets were well-marked. The anterior fontanelle was open, tense and pulsating. The ribs were beaded, the epiphyses of the long bones were enlarged, and the bones of the forearm were curved. On the left upper arm a smooth, firm, tender swelling occupied rather more than the middle third. The swelling felt like periosteal thickening. The right thigh was bent at an angle a little above the centre, and the femur was fractured. The upper fragment was directed forwards and the lower backwards. The lower part of the shaft was much thickened in its whole extent. No crepitus was elicited between the fragments, but there was extreme tenderness over the whole thigh, and the child cried with a shrill, piercing scream when the limb was touched or moved. The temperature was 96° and the pulse 100 per minute, small and weak. There was nothing abnormal in the heart, lungs or abdomen.

On *March* 23 (two days after admission) signs of cerebral affection were present. The child was sick after food or medicine. Pulsation had ceased in the fontanelle, and the pulse was weaker. There was rigidity of the neck. The pupils were dilated, and the head and eyes were directed strongly to the left side. The left side of the face was drawn up. Both arms were rigidly flexed, and the hands were supinated; there was a good deal of tremulous movement in the arms at times. The child sank gradually, and died the same day.

Necropsy.—The body was fairly-well nourished. A subcutaneous hæmorrhage was present over the right frontal region, and another over the front of the right thigh. The skull was markedly rachitic. On opening the dura mater there was found a large amount of blood, dark and fluid, covering the vertex on both sides, from the middle of the frontal to the posterior part of the parietal regions. The surface of the brain was covered with a layer of blood clot, and the cortex was deeply stained. The blood also extended between the hemispheres. The structures at the base of the brain were covered by a thick, yellowish membrane, which was very tough, and contained no tubercles or nodules. It extended about half-way upon each side, and passed into the Sylvian fissures.

The brain contained two hæmorrhagic cavities under the cortex, situated in the posterior frontal region, one on each side. That on the right side was the size of a small Tangerine orange, and contained grumous, sanious fluid. The cavity on the left side was smaller and more irregular, and was partly filled with a small quantity of yellow, turbid fluid. Under the microscope, this fluid was seen to consist of blood-cells, mixed with a considerable number of fattily degenerated corpuscles. The lateral ventricles contained from two to three ounces of clear fluid. The extravasation under the dura mater seemed to be of some standing, from the considerable amount of organisation present in the membrane covering the base of the brain. The arteries at the base of the brain seemed thickened. On microscopic examination, the the basilar artery was found to be occupied by a thrombus which had undergone some degree of organisation.

The right femur was broken across almost transversely immediately above the centre. There was no attempt at union of the fragments, which were considerably separated. A large amount of soft material was lying round the shaft, under the periosteum, for the most part unorganised, but of a cartilaginous consistence in parts. A similar condition was present in the left humerus,

there being no appearance of union between the fragments, and a large quantity of soft material surrounded the broken ends of the shaft. The liver was tough, rather wrinkled and small. The other organs presented no abnormal appearances.

Case 2.—Female child, aged 14 months : rickets ; œdema of eyelids ; anæmia ; cachexia ; hæmorrhages in skin, scalp, and conjunctiva ; ulceration round teeth ; tenderness and œdema of extremities ; syncopal attacks ; death ; autopsy ; hæmatoma of dura mater.

This patient was seen for the first time on November 11, when she was ten months old. She had had a fit five days previously, which had left her with convergent strabismus. Abdominal pain seemed to be present. The nose and hands were blue, and the mother stated that the child was subject to cold extremities. She was wasted and seemed to have been neglected. The skull was large, and there was distinct bossing in the frontal and parietal regions. The anterior fontanelle was widely open, and the sutures had not closed. The heart was normal, and a good deal of bronchitis existed in the lungs. The abdomen was prominent, and both liver and spleen were enlarged. There was no history of hæmaturia. The patient was suffering from "snuffles," but there was no other evidence of syphilis, nor could any history of it be obtained. Her diet at the time consisted of cow's milk.

As regards the family history, both parents were healthy. There had been twelve children in the family, of whom only three survived, the others having died young, one from jaundice, and the rest from bronchitis. There had been no miscarriages.

The patient had always been subject to bronchitis. She never had fits with the exception of the one mentioned. In the end of the following January the child was seen again. Both upper eyelids were œdematous. She was suffering from another attack of bronchitis, and looked ill. She was admitted to hospital on February 10.

On admission, the patient was moderately well nourished, but markedly anæmic, with a yellow translucent skin. Over the right parietal bone, and part of the occipital, was a soft fluctuating swelling, considered hæmatomatous. The head was large and natiform, with distinct bosses. The anterior fontanelle was open, and the wrists were rather thickened. There were two subcutaneous hæmorrhages on the back about the size of a shilling piece, and another on the inner surface of the right arm. There was no œdema of the extremities. The right arm was kept rigid,

and the thumb was firmly adducted; there was also some tendency to adduction of the left thumb. The tongue was furred and dry, and there was some ulceration round the teeth. The liver extended one inch, and the spleen one-and-a-half inches, below the costal margin. The heart was normal, and there was a good deal of bronchitis. The patient was unable to swallow, and had to be fed with the stomach tube. The temperature was 100°, and the pulse 120, rather weak. The urine contained a trace of albumin, but no blood.

On February 12 she had a syncopal attack: the extremities became blue, the pulse could not be felt, and the pupils were contracted. She recovered under stimulation.

On February 15 she seemed to be improving. Her colour was not so yellow. She was still very apathetic, although not unconscious, and cried out whenever she was moved. As a rule she laid perfectly still, moaning at times as if in pain. The thighs were drawn up and adducted.

On March 7 an eruption of hæmorrhagic spots appeared on the abdomen and the back, for the most part small in size. This was accompanied by a rise in temperature, which had been normal for some days, to 101.4°, and by diarrhœa, the motions being green and offensive.

There was another syncopal attack on March 9. On the 13th there were numerous fresh petechial spots over the trunk. The hands and feet were swollen, and the lower limbs seemed very tender to touch. There was a marked increase in the diarrhœa, which had been more or less present during the previous week.

She died on March 26. The child had been lying in a state of torpor since the last note. At times there was a condition of general tonic spasm of the muscles, the eyes being turned to the right. The extremities were blue and œdematous, and the cardiac action was very feeble. The abdomen was extremely retracted. The temperature had been gradually failing, with occasional pyrexial attacks, and for two days before her death it was 95°. No treatment had any effect. The diet consisted of fresh cow's milk, milk puddings, lime juice, and brandy, and medicinally she had grey powder, mercurial inunction and cod liver oil.

Necropsy.—The body was extremely emaciated, with a complete absence of subcutaneous fat. A subconjunctival hæmorrhage was present in the inner and upper surface of the right eye. The teeth and gums were covered with sordes. On the

mucous membrane of the lower lip opposite the central incisors, was an ulcer, deeply excavated, with hard, raised edges, and a yellowish base.

The skin was wrinkled and parchment-like. The anterior surface of the body, from the nipple line to the umbilicus was densely spotted with purple, *ante-mortem* petechiæ, mostly the size of a pin's head. There were a few *ante-mortem* bruise-like marks about the extremities, of larger size than those on the trunk, notably one on the outer side of the right leg, and two symmetrically placed on the back of each forearm, the size of a threepenny-piece.

There was a considerable anasarca of both feet and ankles, and clear fluid escaped on puncturing these regions. A more solid œdema occupied the backs of both hands. There were no ulcers or excoriations about the arms or buttocks.

The skull was typically natiform. The parietal and frontal eminences were very protuberant, and the fontanelle was open and depressed. The epiphyses of the long bones were also somewhat enlarged. Incisions made over the tibiæ showed the bones to be normal. There were no periosteal thickenings, and no subperiosteal hæmorrhages. There was no epiphysitis anywhere in a more advanced condition than that mentioned above.

There was some collapse in both lungs, and pleuritic adhesions were present on the right side. The liver was small, mottled on cut section, and there seemed to be some increase of Glisson's capsule along the portal vessels. The spleen, kidneys and intestines appeared normal.

On dissecting off the scalp, a ragged red appearance was noted on the inner surface covering the right parietal bone. A large hæmatoma had been situated here three weeks before death. The parietal and frontal eminences, and also the squamous portions of the temporal bones, were much thickened, of a deep red colour, and soft enough to be indented easily by the finger-nail.

Beneath the dura mater there was in the situation of the subdural space, a dense, fleshy, purplish red, smooth and soft deposit, which completely covered the surface of the brain. It was generally loosely adherent to the under surface of the dura mater, and could be easily peeled off, except in the neighbourhood of the sinuses, where it was more firmly attached. The dura mater above it was white and glistening. The deposit varied from one-sixteenth to nearly three-quarters of an inch in thickness, the thickest parts being towards the base of the skull in the anterior and posterior fossæ. Beneath this layer, and easily

Case 2.—Vertex of skull shewing false membranes *in situ*. The different layers, with cotton wool pledgets interposed, are seen on the left side of the skull anteriorly.

separable from it, was a second dense white membrane, and on the under surface of this was a third layer of soft, yellowish, coagulated lymph, somewhat closely adherent to it, and to the pia mater beneath. On removing this third covering, the brain was exposed, and presented the following unusual appearances. It was generally atrophied, and weighed fifteen ounces. The convolutions were ill-defined, and narrow, and the sulci were obscured, particularly the fissures of Sylvius and Rolando, by a layer of yellowish solid lymph, encircling the contracted and anæmic blood-vessels, and causing the pia mater to bulge externally.

The convolutions generally were of a pale greyish colour. In striking contrast to these were certain limited convolutions, which were of an entirely different aspect. The latter were of a dirty orange yellow colour, considerably depressed below the general surface of the convolutions, feeling so hard and dry to the touch that they were thought at first to be the seat of old caseating gummata. However, on section, it seemed that only the pia mater and the extreme outer surface of the grey matter were thus affected. The pia mater was thickened and gritty, and perhaps the yellow appearance was due to a fatty degeneration of old inflammatory deposits.

The various cranial nerves were white and glistening, and to all appearance normal. There was no thrombosis of the sinuses, and no disease of the petrous, or any other bone. On section, the brain was found to be tough, but otherwise normal.

On opening the spinal canal, there was a deposit of soft material like red currant jelly beneath the dura mater, and covering the cord, particularly well-marked in the posterior dorsal region. It seemed justifiable to regard this as an effusion of blood-stained lymph, and as perhaps resembling the early stage of the much more advanced effusion in the cranial subdural space.

Microscopic Examination.—The false membranes around the brain presented the appearance of blood clot in different stages of organization. In some parts they consisted of fibrinous tissue containing unchanged blood-cells, in others organization was more advanced, and strands of connective tissue were found, forming spaces which contained groups of altered blood-cells.

In the liver there was a slight increase in the amount of connective tissue surrounding the portal vessels, but the hepatic cells were unaltered.

Remarks on Case 2.—In this case we have a combination of diseases. The signs of rickets and scurvy are, I think, unmistakable, and there is also a certain amount of evidence in favour of syphilis. The distinct bossing of the skull is by some regarded as distinctive of syphilis, while by others, among whom is to be included Dr. Barlow, it is accounted a manifestation of rickets. The question has not been definitely settled, and is not of special importance in this case, for while the possibility of syphilis being present may be admitted, the whole series of symptoms, and the pathological changes found at the *post-mortem* were not such as are present in syphilitic disease, but were quite in accordance with a diagnosis of rickets and scurvy. Further, mercurial treatment had been employed without benefit, both while she was attending as an out-patient, and after her admission to hospital.

Remarks on Cases 1 *and* 2.—In both of these a leading feature was the condition known as pachymeningitis interna hæmorrhagica, or hæmatoma of the dura mater. This was extremely well marked in the latter case, and was present to a slighter extent in the former.

The best account of this affection is given by Huguenin in Ziemssen's "Cyclopædia of the Practice of Medicine" (vol. xii.) He says: "Pachymeningitis occurs in scurvy; here, however, we have no personal observations to offer. Many cases exist where syphilis is supposed to be the original disease; the connection is not clear; certainly no one will be disposed to refer the affection to Heubner's disease of the cerebral vessels."

In Dr. Immerman's article on scurvy in Ziemssen's Cyclopædia (vol. xvii.), which is extremely full and comprehensive, the occurrence of intracranial hæmorrhage is regarded as very exceptional amongst adults. He says: "As very rare local affections of scurvy are to be considered the isolated cases of intermeningeal hæmorrhage, which usually run their course under the form of rapidly-fatal apoplexy, but also in some cases lead to death with the appearance of gradually augmenting compression of the brain, headache,

dulness, and finally stupor ; it is still doubtful whether or not hæmorrhages occur in the substance of the brain." He does not record any case with *post-mortem* examination.

Huguenin refers to Virchow's theory that pachymeningitis is a hæmorrhagic inflammation of the dura mater, but is of opinion that inflammation is never present, and that the whole process consists of extravasation of blood, at first from the dura mater, and subsequently from already existing false membranes.[1] As regards the pia mater, he has frequently seen " the changes attributed to recurring fluxionary hyperæmia (formation of small fibrous callosities, or at times a thick, dense, leathery membrane, in connection with atrophy of the brain)." He has also observed " a yellow colouration of the arachnoid, of the subarachnoid fluid, and even of the surface of the brain, penetrating to a greater or less depth." Amongst all the cases that Huguenin was able to collect, he found that 2·7 per cent. occurred in children under twelve months, and 2·7 per cent. in children from one to ten years of age.

These appearances recorded by Huguenin are very similar to the ones I have described in the two cases under consideration. About the nature of the meningeal changes there can be no doubt ; the only question is as to the primary disease, for hæmatoma of the dura mater is always a secondary affection. In the history of the cases I have adduced the evidence on which I base the diagnosis of scurvy. In hæmatoma of the dura mater we find :

(1) A tendency to recurrent attacks of hæmorrhage without local inflammation.

(2) That coagulation and organization of the extravasated blood occur, and

(3) That the effects are entirely those of mechanical pressure.

These conditions are also characteristic of the subperiosteal and other hæmorrhages of scurvy.

In Case 1 there was no evidence during life as to the

[1] Dr. Wigglesworth also supports the non-inflammatory view. *Vide* BRAIN. Parts lix. and lx., 1892. " Remarks on the Pathology of so-called Pachymeningitis Interna Hæmorrhagica."

period of onset of the meningeal affection, but the organized membrane at the base of the brain pointed to an attack some time previously. In Case 2 the occurrence of a fit at the age of ten months, accompanied by strabismus, points to the first extravasation of blood from the dura mater. Accepting this as our guide, we find that the patient lived four months after the commencement of the pachymeningitis.

OBSERVATIONS ON A CHINESE BRAIN.

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THE following is a description of the external anatomy of a Chinese brain, including notes as to its weight and capacity. It belonged to a Chinaman, aged 36, who was admitted into this asylum last year suffering from general paralysis. Of this the physical signs were the most pronounced. His gait was very weak and unsteady, tongue markedly tremulous, speech very defective, almost abolished, pupils irregular and unequal, knee reflex exaggerated. Mentally he manifested a considerable amount of *bien être*, laughed in a silly, fatuous manner, and at times had many delusions of grandeur. The disease was cut short by the patient dying seven weeks after admission from an attack of pneumonia, and we may fairly take it that there had not been sufficient time for it to produce any marked atrophy of the convolutions which might cause an apparent alteration in their arrangement.

Literature.—The first report on any Chinese brain appears to be that[1] in 1886 by Mills and Parker. Three others[2] were described soon after by Benedict. Dercum in 1889 reported two more,[3] and again in 1892 he published notes on an additional one.[4] As far as possible a comparison will be made between this eighth Chinese brain now under consideration, and the above previously described ones.

[1] "Preliminary Study of a Chinese Brain," by A. J. Parker and C. K. Mills, *Journal of Nervous and Mental Disease*, vol. xiii., No. 10, 1886.

[2] "Drei Chinesen Gehirne," by Prof. Dr. M. Benedikt, *Medicinische Jahrbücher*, 1887.

[3] "A Description of Two Chinese Brains," by F. X. Dercum, *Journal of Nervous and Mental Disease*, July, 1889.

[4] "Note on a Chinese Brain," by F. X. Dercum, *Journal of Nervous and Mental Disease*, September, 1892.

Shape and Size.

Without resorting to the balance, the brain is easily noticed to be considerably smaller than those we are accustomed to see, and to be of a peculiar shape. The difference in shape appears chiefly due to the increase in the greatest transverse diameter as compared with the antero-posterior. On actual measurements of the cranial cavity, the greatest width was found to be 14 cm. (5·5 in.), and the extreme length 15·5 cm. (6·125 in.). The cephalic index is therefore 90, and the skull a markedly brachycephalic one. The hemispheres viewed from above do not display their usual fairly regular oval contour. From the region of the parietal eminences, the point where they attain their greatest width, they rapidly taper towards the extremities, reminding one of the simian cerebrum; this is chiefly noticeable posteriorly, causing the occipital lobes to look small and pointed. From the hinder part of the corpus callosum the hemispheres appear to gradually gape asunder till at the extremities of the occipital lobes they are an inch and a half apart. Looking at the base of the brain, there is none of the eversion of the basal surface of the frontal lobe, noted as being present in all the previously described specimens, but there is well marked eversion of the basi-temporal surface. Benedict noted this in each of his three specimens. In this brain the anterior parts of the temporo-sphenoidal lobes are poorly developed, being thin and wedge-shaped.

The weight of the whole brain is 1,182 grammes (38 oz.), that is, 176 grammes less than the weight of an average male adult brain. This diminution in weight is not shared by each division of the brain, but belongs to the cerebral hemispheres; for these weigh together only 995 grammes, while the average weight of them in a normal European brain is 1,171 grammes. The cerebellum weighs 163·2 grammes, and the pons and medulla together 23·3 grammes, these being just about average weights.

The cubic capacities of the several parts were ascertained by immersing them in graduated glass measures, partially filled with water, and the following figures were obtained :—

Capacity of the hemispheres, 822 cc. (50 c. in.); cerebellum, 141·7 cc.; pons and medulla, 21 cc. Comparing, in man and in apes, the proportion the cerebral hemispheres bear to the cerebellum, in the case of the former it is as $8\frac{1}{2}$ to 1 and $5\frac{3}{4}$ to 1 in the chimpanzee (Huxley). Making a similar calculation in this Chinese brain the proportion is exactly 5 to 1. Supposing that our specimen is fairly typical of a normal brain of that race, and even allowing that there was sufficient time for the disease from which the man was suffering to produce some atrophy, even then there must be a considerable difference in the degree of the relative development of the cerebrum and of the cerebellum to account for the above remarkable change in the proportion.

The Five Fissures.

The Fissure of Sylvius gives off quite normally on each side its small anterior and ascending branches, and continues backwards as usual as the posterior branch. In two of the previous seven brains it is mentioned as being unduly long. In this specimen, as far as the spot where it turns upwards into the tail, it is of normal length; but the tail itself on both sides can be distinctly traced further than usual, especially on the left side. This point will again be referred to under the parietal lobe. On both sides the tail, instead of inclining upwards and backwards, practically forms a right angle with the posterior branch of the fissure, and thus comes really to be directed more forwards than backwards.

The Parieto-Occipital Fissure is well marked on both sides. As regards that part of it on the mesial surface of the hemisphere (the internal parieto-occipital fissure), there is nothing special to notice. Benedict noted that in one of his specimens it did not become on one side confluent with the calcarine fissure. In this brain there is no such abnormality on either side; the calcarine fissure is reached in the usual position, namely, about its middle, on a level with the posterior extremity of the corpus callosum. That portion of the fissure which is situated on the dorsum of the hemisphere

(the external parieto-occipital fissure) is interesting in regard to its length. It makes its appearance at the normal point, midway between the sulcus of Rolando and the occipital extremity of the hemisphere, but is carried to a remarkable extent outwards on both sides, particularly on the right. This part measures generally much less than an inch. In the specimen in hand it extends outwards for an inch and a half on the right side, and an inch on the left. If we take into consideration the general small size of the hemispheres, the length of these fissures would be comparatively greater still. Quain states that the size of the external parieto-occipital fissure depends inversely on the size of the convolution which curves round its outer extremity and connects the parietal with the occipital lobe. And he goes on to say that in consequence of the development in man of this and similar connecting convolutions, this fissure is much less marked in the human brain than in that of the higher apes.

The Calcarine and Hippocampal (or Dentate) Fissures.—In our specimen these two fissures follow a perfectly normal course, except that the former departs somewhat from its usually fairly horizontal direction, its most posterior part being markedly sinuous. On neither side is there the slightest attempt at any confluence between them, the gyrus fornicatus being well developed throughout its whole extent. In each of the three brains described by Dercum, this union took place on both sides; it was present also on both sides in one of Benedict's three cases, and Mills and Parker in their specimen noted that on the right side the gyrus fornicatus became greatly reduced at this spot, the two fissures very nearly becoming continuous.

The Collateral Fissure (Occipito-Temporal) is well marked on both sides, and shows no departure from the normal.

The Frontal Lobe.

The Furrow of Rolando (Central Sulcus) belongs, of course, as much to the parietal as to the frontal lobe, but it is convenient to consider it now. In the previously

described Chinese brains the point, on which stress is laid with regard to it, is the presence or absence of union between it and the Sylvian fissure. In nearly every instance on both sides was this confluence complete or almost complete. It was very nearly so on both sides of the Mills-Parker brain, on the right side of Benedict's first reported specimen, in both hemispheres of his second, on the left side of his third, on the right side of Dercum's first brain, on both sides of his second, and in the left hemisphere of his most recent case. It was freely confluent with the fissure of Sylvius on the right side of Benedict's third specimen, on the left side of Dercum's first, and in the right hemisphere of his latest brain. This apparently regular confluence or approach to it is on neither side observable in the specimen in hand, and certainly on the right side there is a wider loop of grey matter than usual connecting the ascending frontal and parietal convolutions and shutting off the sulcus of Rolando from the Sylvian fissure. The sulcus at its upper end notches the upper margin of both hemispheres, but on neither side is it continued on to their mesial surfaces. Curiously the upper extremity of the right Rolandic furrow is nearly a quarter of an inch behind its fellow; this is seemingly accounted for by the greater length of the superior frontal convolution on the right side. In both hemispheres the sulcus pursues an uninterrupted course.

The Præcentral Sulcus (*Transverse Frontal Furrow*) is not nearly so much broken up as is usual, and is almost as prominent as the central sulcus, and, as we might therefore expect, there is a particularly well developed ascending frontal (anterior central) convolution. The sulcus on neither side is continued completely to the upper edge of the hemisphere, nor is it on either side confluent with the Sylvian fissure, and therefore, at any rate in this case, cannot be regarded as the prolongation of the ascending branch of that fissure.

The Superior, Middle, and Inferior Convolutions are well developed and distinctly mapped out on either side by the two horizontal sulci. They are subdivided by other secondary

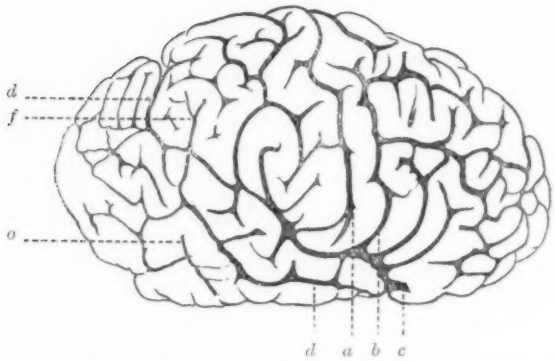

FIG. 1.—Exterior surface of right hemisphere.

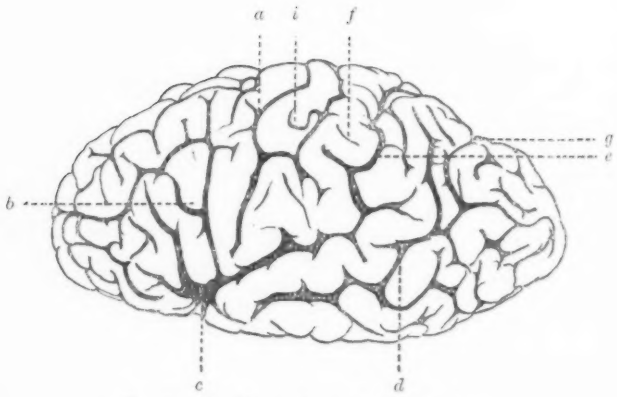

FIG. 2.—Exterior surface of left hemisphere.

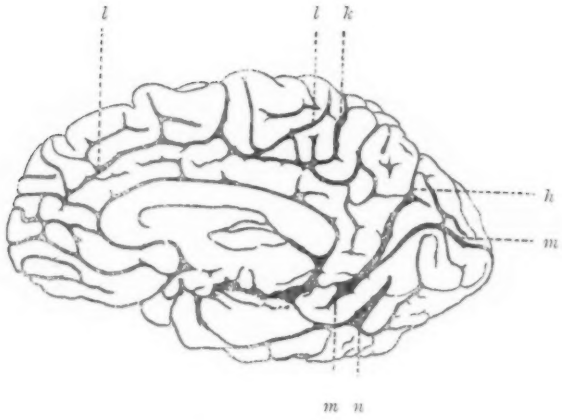

FIG. 3.—Mesial surface of right hemisphere.

a. The sulcus of Rolando.
b. The præcentral sulcus.
c. The Sylvian fissure.
d. The parallel sulcus.
e. Shewing the marked prolongation of the Sylvian fissure on the left side.
f. The external parieto-occipital fissure.
g. Position of the left parieto-occipital fissure.
h. The internal parieto-occipital fissure.
i. The ascending parietal convolution.
j. The provisionally termed posterior ascending parietal convolution.
k. The continuation on to the mesial surface of the post-central sulcus.
l. The calloso-marginal sulcus.
m. The calcarine fissure.
n. The collateral fissure.
o. The middle temporo-sphenoidal convolution.

NOTE.—The upper limit of fig. 2 does not correspond with the upper edge of the hemisphere, but to a line about an inch external to it; hence the marked transverse furrowing is not so well shewn as it might be and the parieto-occipital fissure is not shewn at all.

sulci. The greatest complexity is in the fore part of the frontal lobes; here the gyri are considerably multiplied, and look small and closely packed.

The Orbital Lobule.—The absence of any eversion of this part of the frontal lobe, noted as being present in all the other seven Chinese brains, has been already referred to. The mapping out of its surface into interior, anterior and posterior gyri is not easy; the only one well marked is the internal, and in it on either side is a deep olfactory sulcus.

The Parietal Lobe.

The Intraparietal and Postcentral (Retrocentral) Sulci will be more conveniently studied together. According to the usual description, the former is said to ascend parallel to the sulcus of Rolando for half its extent, and is then directed backwards, blending occasionally with the superior occipital sulcus; and its original direction upward is said to be continued as the postcentral sulcus. On examining the specimen in hand, however, such a description is at once seen to be inapplicable. Here in both hemispheres parallel to the furrow of Rolando is a well marked sulcus, almost as pronounced as the central sulcus itself, in fact more so at its upper end, as it is continued well on to the inner aspect of the hemispheres. On neither side does it open into the fissure of Sylvius. No attempt whatever at any branching from this large transverse furrow is seen on the left side, but in the right hemisphere at the spot where the intraparietal sulcus usually changes its direction, a small branch is given off. Thus in this brain the name postcentral sulcus, besides what is usually meant by that term, would more correctly include the ascending part of the intraparietal furrow. As a result there appears to be in both hemispheres an additional ascending parietal convolution, though on the right side it should be noted that this posterior ascending parietal convolution (if one may so term it) is not so distinct as on the left.

Behind this region the sulci mapping out the remainder of the parietal lobes into other gyri are far from symmetrical

on both sides. In the left one the tail of the fissure of Sylvius is continued upwards to a remarkable extent, practically forming a new transverse furrow taking a course similar to that generally described as belonging to the intraparietal sulcus, for it can easily be followed to within an inch and a half from the upper margin of the hemisphere, and thence is directed backwards at a right angle past the outer extremity of the parieto-occipital fissure and blends with the superior occipital sulcus. In the right hemisphere the configuration of the parietal lobe is more in accordance with the normal. For, as above stated, there is a branch from the large transverse sulcus behind the furrow of Rolando, perhaps representing the hinder part of the intraparietal sulcus; and the tail of the Sylvian fissure can only be said to be continued upwards slightly more than usual. In the previously described Chinese brains there were no variations in the intraparietal sulcus that we are not frequently in the habit of seeing.

The Superior Parietal Lobule.—As follows from the fact that the postcentral sulcus, we saw, was continued on to the inner surface of the hemisphere, on neither side is the upper lobule connected with the true ascending parietal convolution. But in both hemispheres the first annectant gyrus is plainly traceable joining the superior parietal lobule to the upper occipital convolution. On account of the great length of the external parieto-occipital fissure, this annectant gyrus is very sinuous and thin, particularly so on the right side.

The Inferior Parietal Lobule.—Its anterior part termed the supramarginal gyrus appears on the left side to be represented in the lower part of what above has been provisionally called the posterior ascending parietal convolution. That this is so may be gathered from the name given by Turner to this portion of the lobule, namely, the convolution of the parietal eminence, as this part by its protuberance evidently corresponds to the boss of the parietal bone. On the right side, owing to the less complete development of the so called posterior ascending parietal convolution, the supramarginal gyrus is fairly normal. The angular convolution, that is, the hinder part of the inferior parietal lobule, arching over

the upper end of the parallel sulcus, along with the second annectant gyrus, can fairly clearly be traced in both hemispheres.

The Occipital Lobe.

In the three specimens of Dercum, both lobes were observed to be distinctly smaller than normal; especially was this seen in the cuneate lobule. As regards the specimen in hand this fact has been already noted when the general shape and size of the brain were being considered. In the left occipital lobe sulci can be traced dividing it into superior, middle, and inferior convolutions. The third and fourth annectant gyri are seen connecting the last two with the middle and inferior temporo-sphenoidal convolutions respectively. But on the right side three occipital convolutions cannot be made out. The lobe seems divided into two nearly equal parts, the upper, as before said, being joined to the superior parietal lobule by the first annectant gyrus, and the lower has an annectant gyrus connecting it with the inferior temporo-sphenoidal convolution. On neither side is any indication of a transverse occipital sulcus, that is, the small groove said to be generally present in most brains, and directed outwards across the upper part of the occipital lobe a little behind the parieto-occipital fissure. When present it is said to represent the outer portion of the external perpendicular fissure of the ape's brain (*vide* Quain).

The Temporo-Sphenoidal Lobe.

The Parallel or Superior Temporo-Sphenoidal Sulcus is an important one to observe on account of its constancy in all Primates. In the four brains reported by Dercum, Mills, and Parker, attention is drawn to its great length and confluence with numerous perpendicular furrows. In the specimen now studied it is well defined on both sides, and follows very accurately the course of the Sylvian fissure, first backwards, and afterwards being directed perpendicularly upwards, at exactly the same level at which the fissure of Sylvius changes its direction. Its continuation upwards is carried to a remarkable degree on both sides. But there

are none of the transverse confluences mentioned in previous brains observable here, the only junction being that on the left side at the spot where the parallel sulcus changes its direction, the middle occipital furrow opens into it.

Thus a superior temporo-sphenoidal (inframarginal) convolution is well marked on each side. In the right lobe middle and lower temporo-sphenoidal sulci are fairly defined; the former, however, is broken about its centre by a gyrus connecting the middle and lower convolutions together. The middle convolution has no annectant gyrus joining it to the occipital lobe, as is generally the case, but is quite cut off by a small branch from the parallel sulcus first directed backwards, then descending, and ultimately winding into the middle temporo-sphenoidal furrow. Below the inframarginal convolution the left lobe is very irregularly subdivided into small gyri and middle and inferior sulci cannot properly be defined.

The Mesial Surfaces.

The Calloso-Marginal Sulcus.—The usual description given for it applies here in both hemispheres of this specimen, but its termination is not quite symmetrical on the two sides. As is generally the rule, on both sides it ends a short distance behind the upper extremity of the furrow of Rolando, but, while on the right it merely notches the upper margin of the hemisphere, on the left it becomes confluent with the postcentral sulcus. Mills, Parker and Dercum refer to the special complexity of the calloso-marginal sulcus and vegetative repetitions of it were observed by them. None of this is noticed in the present specimen; but in the right hemisphere, as was the case in Dercum's third brain, an apparent addition to the length of the sulcus is seen by its original direction being continued by a furrow in the quadrate lobe also ending by being bent upwards and notching the hemisphere's upper edge.

As regards the convolutions on this surface, no noteworthy peculiarities are observable. The small size of the cuneus has already been referred to, as also has the fact that the gyrus fornicatus is well developed and does not allow of

any attempt at confluence between the calcarine and dentate fissures. The dentate convolution (fascia dentata) was not more pronounced than usual; this fact is noted because in some animals it is much more greatly developed than in man.

Summary.

Before summarizing the above facts it may be well to state here that as regards the markings on the island of Reil and the configuration of the cerebellum, pons and medulla, no departure from the normal is observable.

Although several decided differences from what we look upon as the normal have been pointed out in this and the other seven Chinese brains, the material studied has been too limited to justify general conclusions. But there are certain features which occur with such marked persistency, some of them in all, and others at any rate in the majority of these specimens, that they cannot be regarded as mere coincidences.

First of these stands out the greater prominence given to furrows running transversely as compared to those in the antero-posterior direction. This has been pointed out in every specimen, and is well illustrated in the present case by the three large sulci running parallel with each other in both hemispheres, namely, the central itself with the præ- and postcentral sulci practically as distinct as itself. And in addition to these we have seen on each side an unusually long external parieto-occipital fissure, and that the parallel sulcus was prolonged to a remarkable extent in a transverse direction. This pronounced perpendicular furrowing is still further accentuated on the left side by the undue extension upward of the Sylvian fissure.

Then, as to convolutional complexity, taking the brain as a whole, it is certainly up to the normal standard, and in the frontal lobes even rather beyond the average; this latter point was observed, too, in the first pair of Dercum's cases.

In all his three specimens the occipital lobes, and more especially the cuneate lobules, were noted as not attaining the normal size. This has already been mentioned as being

the case in the present specimen and thus is a point which is constant in at least half the number of brains studied.

If this brain is at all typical of the race to which it belongs, then the small size and weight of the cerebrum as compared to the cerebellum is a point worthy of special emphasis.

It is hoped that the above observations may prove of interest to those who study the comparative anatomy of the human brain, and that as more material accumulates, generalizations of a greater value may be obtained.

ON GENERAL PARALYSIS

(WITH A CRITICAL DIGEST).

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I.

ANATOMY IN RELATION TO GENERAL PARALYSIS OF THE INSANE.

IN order to clear the way for the discussion of the pathological anatomy of general paralysis, it is desirable to speak beforehand of (A) the normal anatomy of the intercellular, or interstitial substance, or stroma, or matrix of the brain, particularly of the brain cortex; and (B), in a subsidiary way, to refer to disputed points concerning the lymph-system of the brain. These are, and are yet to be, battle-grounds of anatomists.

A.—THE INTERSTITIAL, OR INTERCELLULAR, OR CEMENTING SUBSTANCE, OR MATRIX, OR STROMA OF THE BRAIN, OFTEN TERMED NEUROGLIA, BUT PART OF IT, ALONE, RECEIVING THIS NAME FROM SOME.

One must leave aside discussion of the numerous irreconcilable observations and views on this tissue or substance. It has been termed a non-nervous constituent of the brain; has by most (as *e.g.*, Witkowski, Ziegler, Obersteiner), been placed among the connective tissue structures, and has been variously divided. Nevertheless, Robin and Loewe long ago declared it to be nervous matter of a simple kind; and Klebs expressed a similar opinion in speaking of pathological cases. As Gierke stated, the supporting tissue of the central nervous system is comparable with connective tissue in work or functions generally, but essentially distinguishes

itself therefrom by its development, appearance, &c. It has often been described, and usually under the name neuroglia, as being partly of homogeneous, structureless, or, again, finely granular, or molecular, or even fibrillated-looking, material—partly of a structure constituted of fine connective-tissue fibrils, and of cells forming their origin or nodal points—and partly of nuclei, or "free nuclei."

Obviously, its functions are not only to form an imbedding or protective material, but also a supporting structure or scaffolding for the delicate and vulnerable anatomical nervous elements, and for the minute blood-vessels. Less obvious, but now sufficiently clear is also the existence of plasmatic functions of portions of it, in relation to the local lymph-circulation, and hence to brain nutrition and welfare. And, to some extent it may have lowly nervous function, irrespective of that of the definite nerve-fibrils coursing in, or through, it. Just as these several functions vary in relative importance and necessity in different parts of the nervous system, so do we find corresponding differences in the relative preponderance of one or another of the several constituents of this tissue and substance. Thus, in the outmost, or superficial, layer of the cerebral grey cortex is a closely-woven feltwork of connective tissue; whilst throughout the cortex generally the so-called "ground-" or glia-substance (presently to be described) appears to be, relatively, in unusual quantity; on the other hand in the white brain-matter the reticulum of glia-cells and fibrils, with little, if any, ground-substance supports the nerve-fibres; and, again, at the radial septa of the spinal cord, and associated with the large densely-ranked nerve fibres, is a close network of neuroglia cells and fibrils, with but little ground-substance.

To clear the present use of the terms employed, I must enumerate, in detail, the several different materials described as constituting this tissue material, which is disposed between the units of the cortical nerve-cell-and-fibre apparatus, and blood-vessels.

(1) The ground-substance, or cement- or glia-substance; to which constituent alone, some (as, recently, Obersteiner, and Binswanger in places) restrict the name neuroglia—

a name occasionally limited (nominally, but not really, if what I state below is correct), to sustentacular tissue of ectodermal origin[1]—and often applied to the whole tissue and material in question, as sometimes, indeed, to the whole, is the name "ground-substance" itself,—incidental proofs of the enormous discrepancies in nomenclature on this subject.

It has been described, variously, as being unformed, structureless, homogeneous and softish; or by others as being molecular, or finely granular:—

(2) Free glia nuclei, or neuroglia nuclei.

(3) Fibrils and processes; and

(4) Cells, of various types (both "(3)" and "(4)" to be described below).

As to the first of these, "(1)," much of that which was termed glia-, or ground-, or cement-substance has been microscopically resolved into medullated and non-medullated nerve-fibres and fibrils; and the fine granules of neuroglia ("interfibrillar granules" of Boll) attached to processes of nerve-cells and connective tissue, have been stated (Obersteiner) to belong to the nervous system and not to connective tissue.

By some recent methods of investigation, the spaces between the nerve-cells are found to be largely occupied by a host of nerve-fibres and fibrils, formed not only by the collaterals and terminals of nerve-cells, but also by those of fibres coming from the corpus callosum and other distant organs, and by the collaterals of association-fibres.

As to the existence of the nuclei forming the second "(2)" of the above-mentioned constituents of brain-stroma, and as to the morbid appearances attending their asserted proliferative hyperplasia; it is probable that these so-called "nuclei" are, or mostly are, cells in reality; and the appearance explained as being due to a supposed nuclear proliferation, as in general paralysis, has been attributed otherwise (as by Lubimoff) to an effusion of lymphoid elements; or of leucocytes. These "nuclei," may, therefore, be provisionally accepted as cells with little cell-protoplasm, even by those who still look upon them as represent-

[1] As by Ph. Chaslin, *Archives de Méd. Expérimentale, May*, 1891, p. 339.

ing hyperplasia of natural elements of the part. They stain highly with carmine, and some aniline colours.

Of the elements, of the tissue under discussion, enumerated above, there remain, therefore, only the third and fourth, "(3)" and "(4)," as being of paramount importance in either normal or abnormal states; and they will now be dealt with conjointly, *i.e.*, the cells and the processes or fibrils pertaining to them.

To be brief, these cells with their appurtenant processes and fibrils may be divided into two groups.

(*a*) *First group.*—One of them consists of *primary* glia-cells, which are of ectodermal (epiblastic) origin, and under certain aspects or methods of viewing them, have a horny appearance. From some of these proceeds the thick layer of tangential horizontally radiating fibrils, already referred to, in the outermost layer of the cerebral cortex (where also, in parts, are the apical processes of large pyramidal nerve-cells gaining the surface from the deeper cortical layers, as shown by Retzius); whilst the bodies of the glia-cells of this first group also send far down into the cortex, and in rich profusion, long, slender, uniform, unbranched, nearly straight, fragile fibrils; the fibrils from numerous cells form a network by commingling and intercrossing.

At first glance, these cells with their processes have a general aspect slightly like horse-tails. Others of the glia-cells of this group, and found in the white substance of the brain, give off similar fine fibrils, radiating in various directions, so that they have a thistle-down aspect.

As believed by Ramon y Cajal, Binswanger, and others, these cells and their processes are, embryologically, of epithelial source, and independent of the blood-vessels. They are primary glia-cells, or primary cells of the supporting framework or scaffolding of the cortex; and, of the two, are the kind to which the aspect of cornification is limited (Binswanger), although Gierke has attributed this conditional horny look to all glia-cells. The observations of His (1883 to 1890) throw some light on the subject of these cells, as also do those of W. Müller. For example, in the

embryological development of the spinal cord, His found two distinct separate epithelial-cell forms; one of which provides the spongioblasts which serve for the construction of an embryonic frame-tissue or stroma; the other is formed of the germ-cells—becoming neuroblasts—from which the nerve-cells and fibres develop; although, from their origin, the glia-cells and nerve-cells have separate conditions of growth increase and further development. Hence the glia-cells of this first group are epiblastic (or ectodermal) in origin, and, like the nerve-cells and fibres, arise originally from embryonic epithelial elements.

They have primarily a supporting function, forming, as they do, a framework or scaffolding for the nervous elements.

(*b*) *Second group.*—Occurring freely in the brain cortex, the second group consists of glia-cells and their processes—cells which in normal states are flask-shaped, or of more or less stellate aspect (Deiter's cells); are of mesodermal (mesoblastic) origin; and, besides some finer processes, have numerous protoplasmic or dendritic processes, most of which are comparatively short, stout, coarse, and one or several of which are seen to be attached to the perivascular sheath or adventitial tunic of adjacent blood-vessels. When morbidly hypertrophied, as in general paralysis, these cells and processes have the bloated succulent appearance, and the conformation, to which it would be well to restrict the name "spider cells."

Probably mesodermal in origin, [illegible]oint held to be uncertain by Ramon y Cajal, the cells of this group are *secondary* glia-cells; embryologically, are, seemingly, from connective elements which come in with the invading blood-vessels;[1] are intimately associated with those vessels throughout life; seemingly are plasmatic cells or lymph-system elements;[2] provide a lymph-canalicular system between the blood-vessel- and lymph-channels of the cortex

[1] The well-tinting nuclei observed here in the second half of fœtal life, appearing simultaneously with the ingrowth of blood-vessels, and subsequently increasing greatly in number, are taken to be connective tissue cells by Binswanger.

[2] "Lymph-connective elements," or "scavenger cells," of Bevan Lewis.

of the brain ; and, in addition to their well-known connection with the blood-vessels and perivascular (extra-adventitial) lymph-space, and hence indirectly with the epicerebral space, also, it has apparently been made out with likeliness, in recent observations, and as stated by Dr. Andriezen, that their body and coarse processes are surrounded by a system of peri-cellular lymph-spaces.

Obviously, they are not primarily, but only indirectly, sustentacular elements ; apparently they are especially plasmatic, *i.e.*, lymphatic, in function, and are alleged to be the scavenger cells of the brain. As compared with the cells of the preceding first group, they exhibit more of vital phenomena, have little power of resistance, are much more vulnerable, and do not put on the horny appearance.

Nevertheless, some have even recently still described the spider cells as being connective tissue cells (*e.g.*, Obersteiner, 1890).

Dr. Alex. Hill suggests that "neurogleia" (as used by another as equivalent to ground-substance) perhaps partly consists of cell-processes not resolved by the microscope, and partly of coalesced substance of such processes, and that it is not necessary to believe in a non-cellular ground-substance. But he also suggests an application of the terms "neuro-glia" and "connective tissue," differing somewhat from that used in this communication.

Finally, to be consistent with what has been stated above, one could only partly accept the first, and would reject the second, of views recently expressed (as by Dagonet, p. 413-6 *op. cit.*) ; for one could only in part admit (1) that the neuroglia (in the wider sense) is an epithelial tissue, and not a connective tissue ; and one would deny (2) that spider cells (representing glia-cells of the second group hereinbefore described), are with complete accuracy to be called "epithelial cells of the neuroglia."

B.—The Lymph-System of the Brain.

The statements immediately preceding, as to the relations of the plasmatic glia-cells to the blood-vessels and perivascular lymph-space, to the epicerebral space, and to the formation of

a lymph-canalicular system, reveal and explain the vast importance of several conditions and lesions found in general paralysis—namely (with diseased coats of blood-vessels) hyperæmia, even to the dilatation of those vessels, so that sometimes they may even be appressed to the walls of the tunnels which traverse the nervous substance and in which they lie—the hyperplasia of adventitial nuclei, or cellular accumulated heaps in the sub-adventitial and perivascular spaces—the overgrowth, to gross "spider" form, of the second of the groups of glia-cells above described—the adhesions of pia to cortex, occluding parts of the epicerebral space, and closing some of the communications of perivascular lymph-spaces therewith—all of which obviously tend to choke the lymph-circulation of the cortex, and to produce lymph-stasis with all the evils attending it. The importance of lymph-stasis and lymph-circulatory disorder in general paralysis is clearly manifest.

II.

Pathological Anatomy and Pathology of General Paralysis.[1]

It is unnecessary to reproduce the numerous and differing pathological views maintained concerning general paralysis. One need only recall the important part frequently assigned to active cerebral congestion or hyperæmia; by some taken as the proximate determining cause of general paralysis, by others as being an important but secondary phenomenon, by still others as comparatively unimportant; and recall also the conflict of opinion as to whether the essential pathological change in general paralysis is principally and primarily of an inflammatory, or, on the contrary, of a degenerative nature:[2]—as to the former of which, again, opinion was divided on the seat of the inflammation, and

[1] Dr. Otto Binswanger. *Die Pathologische Histologie der Grosshirnrinden-Erkrankung bei der allgemeinen progressiven Paralyse mit besonderer Berücksichtigung der acuten und Frühformen. Jena*, 1893.

Dr. J. Dagonet. *Annales Médico-Psychologiques*, Nov. 9, 1893. Extract from a Treatise on "Mental Diseases" in the Press by Dr. H. Dagonet.

[2] See the writer's "General Paralysis," second edition, pp. 333-4, 338.

its interstitial or parenchymatous character:—and as to the latter of which there was little harmony on the alleged character and course of the degenerative process.

Some use of Dagonet's *résumé* has been made in part of what immediately follows.

Most of the earlier observers of general paralysis took it to be a chronic meningitis, or chronic meningo-encephalitis or peri-encephalitis; conditions of which the cerebro-meningeal adhesions afforded notable indications, although the frequency of these was far under-estimated.

Then, the atrophy of brain following (as supposed) the obvious meningeal changes, led to the statement that general paralysis was a precocious senile atrophy of the brain, following chronic meningitis (Duchek).

Rokitansky led the way in attributing an important part to hypertrophic changes of the interstitial tissue in general paralysis; the hypertrophy, or the proliferation of which tissue, occurring especially in certain parts of the cerebral cortex, he believed to be produced more usually by hyperæmia, but sometimes by an encephalic inflammation proceeding with the meningitis; and he maintained that this increase of connective tissue had the result of disorganizing the brain-cortex, the nerve-fibres and nerve-cells becoming profoundly altered and broken up. By some, a degeneration of the blood-vessel walls was held to occasion [or promote] the disorders of local nutrition and circulation, and the new formation of connective tissue was described around the blood-vessels of brain and pia. By the increase and proliferation of interstitial tissue, leading to destruction of parenchymatous nervous elements, the lesion of general paralysis was held to be brought into line, and to rank, with a cirrhosis of liver or of kidney. And, although he termed them lymph-connective elements, possessing the function of brain-scavengers—a function overwrought in general paralysis, and leading to the well-known overgrowth of the glia-cells of the second group already described, which may then more fitly be named "spider cells"—Bevan Lewis ascribed to them, in this latter state, an aggressive disintegrative effect on the adjoining nervous elements—a view

which falls essentially into line with the above cognate view as to primary connective interstitial lesions in general paralysis, and consecutive dependent parenchymatous ones.

Yet in reality the matter was not so simple as a primary interstitial change, and resulting secondary destruction of nervous elements. For the lesions of general paralysis are complexly associated; no one of them, alone, has pathognomonic character; and general paralysis not being always one and the same, these lesions are differently grouped in separate cases; may be unequally distributed, patches of marked change, whether chiefly vascular, or interstitial, or nervous, perhaps appearing beside comparatively normal structures. And of the several individual tissues constituting the brain, the lesions may vary much in intensity between themselves, for marked parenchymatous lesions may accompany slight interstitial or vascular ones, and *vice versâ*; for example, where the nerve-cell degeneration and destruction are extreme, there may occasionally be scarcely any glia-cell overgrowth.

The blood-vessels and the stroma having thus successively been taken as the chief and primary seat of morbid action, the nervous elements in their turn were credited with playing a similar part; and this in recent years especially, although Meschede had long ago declared the pathological basis of general paralysis to be parenchymatous inflammation of the inner layer of the cerebral grey cortex, and principally affecting the ganglionic nerve-*cells*.

Disappearance of medullated nerve-*fibres* in definite local portions of the cerebral cortex was found by Tuczek. This he was inclined to look upon as a primary lesion; for while leptomeningitis and interstitial encephalitis usually co-exist with the nerve-fibre lesion, this last seemed to be independent, at least in some cases. Those which disappear are the finest fibres. This lesion varies in degree in different cases and in different parts of the brain, affects certain portions of it earliest and most, and there is some evidence of a successive order of implication of the several parts affected. Other observers (Zacher, Greppin, Kronthal, Schütz, Wille, Friedmann, Meyer, Lissauer) have considerably modified the observations

of Tuczek, whilst confirming them in a general way, and have shown that other parts of the encephalon are also often similarly affected, including the cerebellum and optic thalami, where many nerve-cells and fibres may disappear. And the conclusion gained ground that there is a *primary* process of destruction of these nerve-fibres in general paralysis, to which any sclerosis or neurogliar proliferation found is *secondary* (*e.g.*, Strümpell); although a somewhat different and diffuse primary disappearance of nerve-fibres is also observed in some other diseases of the brain. Moreover, it was asserted by Weigert that in tabes dorsalis the nervous elements similarly first undergo degeneration, that the tabetic sclerosis also found is only secondary thereto, and that the same order of events occurs in all cases of proliferation of neuroglia. In general paralysis, Schütz likewise observed destruction and disappearance of the finest fibres of the grey central ventricular nervous substance, which, from a developmental point of view, are stated to form a system, and are, in likelihood, fibres of association between the different cells of the central nuclei of nerves, and so conduce to the synergy of those nuclei. On that basis, this lesion would be systemic or systematic, and rank analogously to the systematic columnar lesions of the spinal cord. The relatively marked change of the nerve-cells and their processes, as compared with other elements, and often observed in general paralysis, is also emphasised by Dr. E. C. Carter.

To establish the atrophy of nervous elements as a *primary* affair, to which any vascular and neurogliar changes found are secondary or complicating, would be to revolutionise the antecedently predominant conception of the pathological processes of general paralysis. For, with (*a*) the general conception of a primary overgrowth of stromal connective tissue or lymph apparatus, whether of inflammatory or of other origin, aggressively producing *secondary* degeneration and atrophy, or destruction and disappearance, of the parenchymatous nervous elements—contrast (*b*) the contrary view of a *primary* degeneration, atrophy and disappearance of nervous tissue; other lesions

present being secondary (or accidental); so that, to refer to the extreme pole of this opinion, Wernicke even held the huge spider cell overgrowth as being passive and compensatory, to fill the void left by the vanished nervous structures.

Some time before I first knew (1885) of Tuczek's results, through his work published in 1884, I held the view that, in some cases at least of general paralysis, the nerve-cell-and-fibre apparatus of the brain probably suffers earliest, and I maintained this view in a paper read at the annual meeting of the British Medical Association in 1883, and reproduced some of the conclusions drawn therein in my work on "General Paralysis,"[1] from which the following extracts are taken, the parts which more especially bear on the point at issue, being italicised now, but not in the original.

"It must be admitted that in many cases of general paralysis, all one sees may fairly be explained without calling in inflammation as a factor; and that, besides hyperæmia or its traces, and atrophy and degradation of true [nervous] elements, some substitution of a texture or substance of altered and lowered type, vital character, degree of development, and differentiation; for and in place of the usual texture of the part. . . . is in some cases practically what we see."

"Then if we enquire where the starting-point of the change is in general paralysis, our histological investigations would, on the whole, refer us[2] to the vessels and interstitial tissue, or at least intercellular elements. *Nevertheless, it may well be that the starting-point of the trouble is often in the ganglionic nerve-cells*, and that the excessive action, over-strain, and morbid stimulation or irritation by which the nerve-cells are brought into an invisible[2] state of damage, injury, lowered vitality, . . . superinduces. . . . active afflux of blood. . . . An equal degree of departure from a healthy condition existing in nerve-cell, in vessel, and in intercellular substance, the last two would give vastly

[1] "General Paralysis," second edition, 1886, p. 340-342.

[2] *I.e.*, by the microscopical methods chiefly in vogue before then.

more obvious histological indications of it than the first would. In the nerve-cell is an excessively delicate organisation. . . . Thus then, the effects of severe disturbance, and the resulting comparatively gross morbid change will first be *visible* in the vessels and interstitial (intercellular) substance."

"On the whole, we may view general paralysis as essentially commencing with hyperæmia, and ending with chronic cortical degenerative cerebritis, and, usually, embryonic- and connective-tissue substitution, the change, *fundamentally parenchymatous*, affecting all the elements of the part, but usually under the methods of examination *hitherto chiefly in use*, presenting a more obvious and more marked affection of the blood-vessel walls and of the interstitial elements. . . . The cerebral, or even encephalic, are almost invariably associated with lesions of other parts of the cerebro-spinal nervous system; and the morbid action begins exceptionally in parts other than the cortex cerebri. Obviously this view allows of several varieties of general paralysis."

And it will not do to make too much of one factor in the morbid histology of general paralysis; and we have satisfaction in noting that Dagonet also recognises both inflammatory and sclerotic lesions as well as the degenerative and atrophic. Besides a vulnerability or selection of nervous elements in it, there is also a vulnerability or selection of blood-vessel walls; the circulatory troubles and lymphatic stasis tell for much, and one must restate that in cases of general paralysis, taken collectively, the lesions are multiple, complexly associated, and may earlier and more affect certain of the tissues composing the brain in some cases or parts, and others of them in others. May we then hope to divide general paralysis into varieties on this particular, and chiefly microscopical, anatomico-pathological basis? There are some indications that such may be the case, or that at least some light may thus be shed on the subject. In my book on "General Paralysis," besides what is said as to alcohol, syphilis, and lead, &c., is reference to a toxic origin as being probable in some cases; and in my article on general paralysis

in the "Dictionary of Psychological Medicine," vol. i., p. 539, it is suggested whether the *primary* destructive change and disappearance of cortical nerve-fibres in general paralysis, and especially associated with a history of drink, "may be due to a form of alcoholic nerve-degeneration or neuritis, akin to alcoholic peripheral neuritis;" by which I mean that alcohol (and other toxic agents) probably may occasion primary nerve-fibre degeneration, or neuritis, not only in the peripheral nerves, but also in the nerve-fibres at the other peripheral pole of the nervous system, namely, at the periphery of the encephalic mass—the brain.

Some, especially recent, observations and conclusions seem to give support to the general view a moment ago suggested.

For example, long ago I published cases which, especially now, would be termed syphilitic general paralysis, or pseudo-general paralysis, in which the smaller blood-vessels (especially arterioles) of the cerebral cortex, were earlier and more markedly affected than the other elements.

The results of Bevan Lewis[1] may be of some value on this point, as assisting to indicate cases of general paralysis in which alcohol is one, or the chief factor; namely, that in chronic alcoholism, as compared with general paralysis, there is a somewhat different situation of the points of chief incidence of spider-cell development; and in the blood-vessel the morbid change is centred in the atheromatous state of the inner coat, the outer or adventitial investment not showing the enormous degree of nuclear proliferation found in general paralysis.

Raymond[2] also finds syphilitic general paralysis to be a chronic interstitial inflammation, having its point of departure in the capillaries of the brain, and more particularly of the cerebral cortex. Following the nomenclature of Gilbert and Lion, the infiltration by embryonic cells leads him to designate as diffuse embryonic meningo-encephalitis; or

[1] "A Text Book of Mental Diseases," 1889, p. 538.

[2] *Archives de Neurologie*, Jan., 1894.

again, he names as diffuse vascular meningo-encephalitis; the brain-lesion of at least syphilitic general paralysis, if not of all forms. In the cortex, and less so elsewhere, the lymphatic sheath of the capillaries, and the lymphatic sheath and the wall of the larger vessels, show infiltrating accumulations of cells with rounded nuclei (nodular or diffuse periarteritis, ordinary secondary obliterative endarteritis; in outer—musculo-connective—venous walls, similar diffuse or nodular infiltrations).

This, with considerable disappearance of tangential fibres, appears to be the earliest change; meningitis co-exists; sometimes with miliary gummata, and often change of the large blood-vessels at the base of the brain, or branching therefrom.

R. Collela[1] concludes that, in the brain:—

(1) In general progressive paralysis with syphilitic infection, the histological changes chiefly affect the blood-vessels; the neurogliar cells [increased size and number of spider cells]; and the cellular protoplasm and protoplasmic processes of the nervous elements. The axis-cylinder processes [and nerve-fibres] are not destroyed, except in a few elements, and only later on. The changes begin essentially in the vascular network.

(2) In paralytic dementia with alcholic intoxication, we plainly observe a hypertrophy [but no notable increase in number] of spider cells; and different phases of regressive nutritive disturbances in the nervous (axis-cylinder) processes. Blood-vessels healthy.

(3) In alcoholic insanity the alteration is essentially parenchymatous, affecting the nervous processes, and with a scarcely appreciable implication of the ganglionic bodies and protoplasmic processes of nerve-cells. Neuroglia and vessels healthy.

At all events, however this may be, further research on this line promises to be more fruitful than a premature attempt to distinguish true general paralysis from the various forms of so-called "pseudo-general paralysis" by fine micro-

[1] From Golgi's Laboratory. *C. R. Acad. des Sci.*, Feb., 1893.

scopical points alone. Thus, we find recent observers who speak of the pseudo-general paralyses as being analagous or else identical; or as being almost identical; *clinically*, with true general paralysis, and sometimes only distinguishable from it microscopically; proceeding to set up as a criterion that true general paralysis, as shown by its vascular lesions, is, rather, an inflammatory affection, in the cerebral cortex at the least—a true encephalitis—and by this alone distinguishable from the pseudo-general paralyses; inasmuch as in these, on the contrary, the lesions are degenerative and regressive.

But when we turn to their account of the microscopical changes in true general paralysis, we find an account (and a correct one) of degenerative alterations in the cortical nerve-cells, proliferation of small round cells in the pericellular spaces, destructive changes in cortical and convolutional nerve-fibres. These changes give no active support to the criterion put forward. But it is the vascular lesions on which reliance is placed for this purpose, and these lesions are stated to be inflammatory and not degenerative; the new formation of vessels is maintained as a fact; and considerable diapedesis in the lymphatic sheath is said to cover or conceal the middle coat. On the other hand, degeneration of muscular coat and large yellow granulations are declared absent. So also is atheroma, if the true general paralysis is not "associated," that is to say, unless its lesions and those of pseudo-general paralysis co-exist.

Now, we shall see presently that vascular degeneration may exist even as an early change in true general paralysis, and thus the alleged criterion appears to fail.

At and from this point, so far as concerns this part of the subject, namely, the cerebral pathological anatomy, we take up exclusively the recent work of Professor Binswanger on the pathological histology of the disease of the cerebral cortex in general paralysis, especially in acute and early forms thereof.

Speaking generally, two series of chronic processes are observed in general paralysis:—

(1) Degenerative—atrophic changes in the specific nerve-tissue.

(2) Swelling, growth, and shrinking in the vascularized sustentacular apparatus.

As regards its lesions, the ground-substance takes a position intermediate to the two just mentioned, inasmuch as it shows both atrophic-degenerative and proliferative phenomena in the advanced forms of general paralysis.

Inflammatory processes in the narrower sense (exudative processes) develop after tissue lesions which call forth reparative processes of new formation of tissue from fixed cell-elements (not leucocytes).

Studying the acute and early forms of general paralysis, he discusses, in order, the several constituents of brain.

Nerve-fibres and nerve-cells.—The typical cases of general paralysis derive from chronic diffuse atrophic degenerative changes of the functionally active cortical substance (nervous elements). Probably these changes first affect the finest terminal ramifications of the medullated and non-medullated fibres (not by our present available means demonstrable with sufficient clearness); but even in the commencing stage of the disease these changes pass over to the medullated nerve-fibres (demonstrable by the staining of the medullary sheath), and to the ganglionic nerve-cells. How far structureless and structural elements of the original (ecto-dormal) ground- and supporting-substance take part in the disappearance of tissue cannot be shown with certainty: only at the nuclei of the glia-cells are the signs of degenerative processes recognisable.

Blood-vessels: distension with red blood corpuscles, hyaline degeneration, reparative thickening of tunics, growth of endothelial nuclei, narrowing of vessel-lumen.—Already in the early stages are the resultant phenomena of these tissue lesions clearly expressed in the blood-vessels. The venous channels are throughout much dilated, distended with red blood corpuscles, while there is not an essential increase of the leucocytes within these vessels. Already, also, does the vessel wall succumb to early regressive alterations, which particularly affect the arterioles and capillaries, and have the character of hyaline degeneration.

At the same time active reparative processes occur at the

endothelial-adventitia of the vessels—beginning at the veins—processes which consist of a thickening of these tunics and increase of the endothelial nuclei. A part of these new formations of tissue also undergoes hyaline degeneration in the later stages of the disease. In other vessels, the proliferative processes attack the true vessel wall later on, and bring about its streaky thickenings and nuclear increase. Thereby the lumen of the vessels is narrowed, but complete obliteration of vessels—as observed in large numbers in old cases dying in the final period—is not demonstrable in the early stages. Neither in recent nor in long-standing cases could a new formation of vessels be established with certainty.

Lymph channel system (of blood-vessels).—In the early stages the system of intra- and extra-adventitial lymph-ways is already extremely dilated.

(*a*) Ampulla-like expansions of the *intra*-adventitial lymph space contain varying quantities of red and white blood corpuscles, coagulated lymph, amorphous, clumpy and finely granular blood pigment. Only few collections of leucocytes within the adventitial sheath are found in the early stages. Extravasation of constituents of the blood, particularly of red hæmacytes, as well as the relics of former such effusions, occur the most frequently in cases presenting wide-spread degeneration of the vascular walls.

In those early forms of general paralysis which take a slow course, uninterrupted by "paralytic attacks," until death happens from intercurrent disease; namely cases which constitute his first group; the accumulation of leucocytes in the endothelial sheath never reaches such a high degree and general spread as to justify the conclusion that exudative processes had existed during life. On the contrary, those leucocytal crowds are only to be taken as results of long-lasting and oft-repeated venous stases.

(*b*) Dilatation obtains throughout the *extra*-adventitial lymph-way network, including here the pericellular spaces. The contents of the extra-adventitial spaces are found to be much the same as, though less in amount than, those of the intra-adventitial already mentioned. An essential increase

of lymphoid elements is not established for the lymph spaces of the cortex and the pericellular.

Stroma : scaffold- or interstitial-substance.—As far as is recognisable by the present methods of investigation, there are not processes of growth or increase of the fibrous parts of the interstitial framework. In the commencing stage it is only in the first or outermost layer of the cortex that (beside atrophic patches) there are evinced distinct increase and firmer quality of the glia-fibres. In the early forms of the disease the mesodermal supporting cells (spider cells) are not increased in the middle and deeper cortical layers. A distinct increase of these cells first appears in the most superficial cortical stratum.

Inflammation, paralytic attacks.—In the later course of general paralysis, and supervening on the changes already named, are true inflammatory, that is to say, exudative processes; which not seldom manifest themselves clinically by febrile action, states of somnolence, phenomena of cortico-motor irritation and paralysis ("paralytic attacks"). Should death follow in such an acute seizure, there are extensive small-cell accumulations in the intra- and extra-adventitial lymph way system. Yet must it be noted here that by no means all so-called paralytic attacks have this pathologico-anatomical basis; much rather may we hold that fluctuations in brain pressure, alone, represent the factors of paralytic attacks—fluctuations which are called forth by the hindered outflow of the lymphatic fluid overfilling the lymph channels of the brain. This will happen the more easily the more intensely the cortical functions are damaged by the diffuse disappearance of (nervous) tissue.

Pia mater; adhesions. Lymph stasis.—Participation of the pia in the morbid process plays an important part in the further development of general paralysis. In the early forms of that disease the pia is thickened and beset with numerous endothelial nuclei over the cerebral convexity, but only slightly so, and limited to isolated situations on the frontal lobe.

But the thickening and nuclear increase of the endothelial-adventitia, already mentioned, regularly appear in the

vessels radiating into the cortex. . . . Both in the sphere of the vessels permeating the cortex and in the thickened pia, occur partial obliterations of the epicerebral space by partial cohesions of the pia with the cortex. These cohesions are produced, on the one side by ingrowth of the glia fibres into the pia; and on the other side by the spreading and growth of the endothelial septa, which, springing from the pia, traverse the epicerebral space. By the cohesion of the endothelial-adventitia with the neighbouring cortex-surface, those apertures of the extra-vascular lymph canal system which subserve outflow are closed, and thereby is produced an increased engorgement with fluid, particularly in the lymph spaces of the outermost cortical layer, but also found in the more deeply situated parts of the cortex. The transformation of perished tissue-materials to coagulated necrotic masses is wrought by the pathologically increased and altered lymph fluid.

Acute galloping general paralysis.—The so-called acute galloping forms of general paralysis are not distinguished from the typical form by any especial necroscopic condition. They are distinguished only by a more rapid progress of the pathological processes. The degenerative as well as the reparative processes gain a quicker and more diffused propagation. In some of the cases death occurs on the basis of the clinical phenomena conditionated by the tempestuous course of the disease; in which violent motor excitation, defective food-taking, exhaustion, broncho-pneumonia, intestinal catarrh, &c., play the chief part. True exudative inflammatory phenomena may then be entirely absent. The same conditions occur more markedly in another set of cases.

Whether it is the typical case of chronic course, or the galloping example of general paralysis, the diffuse pathological process first and chiefly affects the frontal lobe (including the insula) and parietal lobe, and then invades the temporo-sphenoidal and occipital regions. Early extensive participation of the occipital lobe has not been observed by him.

III.

Some Other Recent Studies in General Paralysis.

A.—The Spinal Lesions in General Paralysis and in Tabes; a Comparative Study.

Some have held that the spinal disease in general paralysis and tabes is one and the same; others have held them not to be identical. Joffroy maintained that both general paralysis and tabes are of parenchymatous origin, but that the former is not a "systematic" disease, whereas the latter is so, and of the sensory system. Like some others of us, Marie[1] finds that general paralysis rarely supervenes on tabes; a more frequent occurrence or sequence is the appearance of symptoms of lesion of the posterior columns, either simultaneously with, or in the course of, general paralysis.

Irrespectively of the question of precedence, also, the statistics of G. Renaud show only about 3 *per cent.* of general paralytics with clear symptoms of tabes; about 2 *per cent.* additional presenting more or less pronounced signs of sclerosis of the posterior columns, but insufficient for the diagnosis of tabes.

Marie claims that he had shown, in a previous communication, the differences in site between the lesions of the posterior columns in pellagra and in incipient tabes; in the former (pellagra) the posterior internal zone and the comma-shaped bundle chiefly suffering; whereas in the latter the parts mainly affected are the posterior nerve-roots, Lissauer's tract, the "cornu-radicular" zone and the nervous network of Clarke's columns. Therefore, he terms the former of "endogenous" origin; the latter of "exogenous"; the one change starting within, the other without, the cord.

Making, on the same lines, a comparative study of the lesions of the posterior columns in general paralysis and tabes, and using the figures published by Westphal as a representative test of the leading type of posterior-column change in general paralysis, he finds that the "cornu-radicular" zone is *not* altered (it is the external part of Burdach's column abutting on the middle third of the internal border of the posterior horn, and represents approximately the posterior root-zone). On the other hand, in the posterior columns, the comma-shaped fillet is affected: and so,

[1] *Gazette des Hôpitaux*, January 16, 1894, p. 55.

also, is the "median zone of Flechsig," bordering on the posterior median fissure. The change is poliomyelitic, and is of "endogenous" origin.

These same parts of the posterior-external and posterior-median columns (altered as described in general paralysis with this first type of spinal change), are also affected with descending secondary degeneration in transverse lesions of the grey substance of the cord.

The source of these alterations of the posterior columns in general paralysis he seeks in a poliomyelitis, affecting the so-called "cells of the posterior columns," situated in the grey medullary substance, and described by several recent observers.

But, besides the affection of the posterior columns, there is, in general paralysis, usually, also, a sclerosis of the lateral columns of the cord. He thinks this lesion is not analogous to the secondary degeneration of hemiplegiacs, inasmuch as: (*a*) it is not wholly confined to the crossed pyramidal tract, but invades the neighbouring parts; as (*b*) in some cases the lesion of the crossed pyramidal tract ceases at the level of the medulla oblongata, and cannot be traced above; and as (*c*) the direct pyramidal tract is not similarly lesed.

Various views have been held as to why the crossed pyramidal tracts should degenerate in general paralysis; and Marie holds that the reason is to be sought in the centres on which they depend; that the pyramidal degeneration in general paralysis is analogous to that of the same territory in amyotrophic lateral sclerosis; and that this degeneration of the crossed pyramidal tracts (except for the greater or less part which a morbid cerebral process may or may not take therein) is chiefly produced by the alteration of certain cells, situate in the grey medullary substance of the cord, namely, the so-called "cells of the lateral column." On this view, the degeneration of lateral columns in general paralysis would be chiefly due to a poliomyelitis affecting certain special elements of the grey.

Thus, in general paralysis, two groups of lesions in the posterior and lateral columns, or combined degenerations or scleroses of both columns, are declared to be dependent upon systematised poliomyelitic changes.

Nevertheless, this combined type is not the only one in general paralysis. The lateral columns alone may be affected, or the posterior columns only.

If the posterior columns only are affected, the lesions are much more like those of typical tabes in situation; *e.g.*, the

"cornu-radicular" zone is affected, and the pathological process is "exogenous."

These cases are different from those of the usual type above-described in general paralysis, not only anatomico-pathologically, but also clinically; at least, those are so in which a lesion of the posterior column has for a long while preceded the cerebral symptoms. And, as regards cases such as these last, Fürstner, like others of us, has observed, and has emphasised, the protracted course of the disease; the prolonged retention of mental integrity, and, later on, the unusual degree of preservation of considerable mental power; the comparatively slight affections of speech, tongue and face; the relative rarity of "paralytic attacks"; and he questioned whether these are really examples of general paralysis. Especially in some instances is it difficult to classify certain of the cases of this group, namely, those with the symptoms of tabes still rudimentary only, even after several years' duration, there being merely reflex iridoplegia, absent knee-jerk, hypalgesia, slight bladder trouble; and, on the mental side, only a moderate diminution of memory, some isolated indications of mental deterioration, and a state of apathy. Therefore, overt tabes does not readily turn into general paralysis.

To sum up, then, there are two main great distinct types; one commencing within the cord and poliomyelitic in origin; the other commencing without the cord, and invading by way of the posterior nerve-roots.

Finally, these two types, limned above, may be combined in the same patient, some of the lesions of the posterior columns in such a case being "exogenous," and others "endogenous,"[1] and associated with the lateral column lesion of the first type.

On this view, the spinal and other lesions of general paralysis would be parenchymatous in origin; the toxic agent producing them acting primarily on nerve-cells; namely, on the nerve-cells of the brain, on the nerve-cells of the grey matter of the cord for the so-called "endogenous" changes of the cord, and on the nerve-cells of the spinal ganglia for its "exogenous" changes.

In reference to the foregoing, I think Dr. Marie presents the matter in too schematic a way, limits his view far too much to certain types of lesion, and does not adequately acknowledge the effect of the cerebral lesions of general paralysis in occasioning secondary spinal degeneration, nor sufficiently allow for the great variety, and often diffuse character, or irregular distribution, of

[1] Using these words in the sense already described.

spinal changes in general paralysis. (See the present writer's work on the subject; *op. cit.*, pp. 288 and 322.)

Moreover, the two cases reported by Westphal, and taken by Marie as crucial examples of the lesions of the posterior columns in two different groups of cases forming types of spinal lesions in general paralysis, and occupying different situations in those columns; and the one of "endogenous," the other of "exogenous" origin; differed comparatively little as concerns spinal symptoms; for none of them were ataxic, and the loss of knee-jerk was the one spinal feature in common, and from it alone was posterior columnar change diagnosed.

Nor does either of Westphal's cases, chosen as typical, correspond closely in posterior columnar change with the other case in the same paper, or with the case depicted in a subsequent paper by the same observer in the same publication. This last, indeed, resembles more the tabetic type of distribution of chief spinal change.

The presence or absence of ataxy in cases of posterior spinal lesions in general paralysis, Westphal himself was inclined to make dependent on the intensity of the disease (the degree of atrophy), and on the greater or less participation of the nerve-roots therein. And he laid some stress on the fact that those lesions as found in general paralysis are often unaccompanied by marked, or by any, motor or sensory symptoms.

B.—Spinal Forms of General Paralysis.

It has been widely recognised that some cases of general paralysis begin, or appear to begin, with spinal symptoms, the onset seeming to be by the spinal cord or spinal nerves. Such cases are often called examples of the spinal form, or forms, of general paralysis. General paralysis tends to affect the whole nervous system, but its earlier incidence may be on this or that part of it; and may occasion clinical appearances which call to mind those of various diseases of the spinal cord.

A study of the spinal lesions and symptoms in, and spinal forms of, general paralysis, has just been published by Dr. M. Klippel.[1] As space is limited and time presses, the spinal clinical forms will be briefly noticed, and then the lesions.

The spinal clinical forms of general paralysis (in the above sense) are multiple: some take a tabic aspect, sometimes the early symptoms are those of bulbar paralysis, of lateral sclerosis, of

[1] *Archives de Médecin Expérimentale*, t. vi., Jan., 1894.

muscular myelopathic atrophy, &c. These symptoms are determined by localisation of the lesion more particularly on this or that system or part of the cord, or bulb; and, for purposes of description, the forms must necessarily be more individualised, and less mixed or "combined," than they actually are in fact. Owing to this mixture or association of lesions, the special symptoms do not usually correspond directly in degree to the degree of lesion producing each; the clinical manifestation of the lesion in this part being perhaps modified or neutralised more or less by the lesion in that.

Cases of spinal disease, which, established as such, afterwards become *complicated* with general paralysis, are not included here.

Klippel prefers to divide the spinal, into five forms:—

(1) *The tabetic form.*—With the sclerosis of the posterior columns there is usually also that of the lateral columns. He says the symptoms are, in a word, those of tabes. But, in fact, the symptoms are often only those of an incomplete, or partial, or rudimentary tabes, or tabetiform state.

Besides this form, and to be distinguished from it, are the cases of tabes with cerebral lesions (Jendrassik), but only analagous to, not identical with, those of general paralysis (Klippel). Also, established tabes, late and slowly becoming complicated with true general paralysis, must be kept separate from the form we are considering.

(2) *The lateral sclerosis form.*—Whereas in the first form the lesions chiefly affect sensory elements or conductors of the cord; in this second form the chief incidence is on the motor paths.

Bi-lateral pareses, more on one side; some appearances of muscular contracture, and of exaggeration of reflexes, are the main symptoms; but often are transitory only; the invasion reaching other parts of the cord. Posterior sclerosis is usually conjoined with the lateral.

(3) *The amyotrophic form.*—The state precedent to, and forming the onset of, general paralysis in this form takes either: (*a*) that of progressive muscular atrophy of the Aran-Duchenne type, or (*b*) that of amyotrophic lateral sclerosis (lesion of lateral columns [part of], also of anterior horns, and peripheral nerves).

(4) *Bulbar forms.*—In one case he observed symptoms of bulbar paralysis in a patient whose mind did not seem to be affected, but who three months afterwards showed dementia and the usual indications of general paralysis.

(5) *Neuritic forms.*—As, for instance, neuritis of the limbs with muscular atrophy; especially forming the mode of onset of some cases of traumatic general paralysis.

These are convenient divisions, but do not seem to be absolutely necessary, if we bear in mind the principle involved in them, which has been much debated, but accepted by many.

For the most part, the spinal lesions in general paralysis are situated in some part, or parts, of the posterior half of a transverse section of the cord. They are, in a certain degree, diffuse; and yet present some character of systematisation, affecting, as they do, the sensory or motor elements or both, of a physiological system.

In the posterior columns, there may be only simple thickening of the connective framework, leading by compression to simple atrophy without degeneration of the nerve-fibres; in other cases amidst the sclerous, or seemingly œdematous, tissue are nerve-tubes with granular degeneration of the myeline sheath; in other cases, or in parts of the lesion, the destruction of the nervous elements is extreme. Differences in the site of the lesions present themselves in different cases. The blood-vessels of the affected parts are dilated and are gorged with blood.

Much the same description applies to the lateral columns; and the sclerosis here is not directly as that of the posterior columns (when they co-exist). And often the direct pyramidal tract is exempt, as, to a large degree, also, the rest of the anterior columns usually is.

In the grey matter of the cord are often important lesions of the ganglion nerve-cells and of the nerve-network. Those of the cells may be simple atrophy or degeneration, and in some cases even disappearance of the cells. The cells of Clarke's column may be similarly affected. There may be destruction of the nervous network of the grey substance and Clarke's column.

In the nerve roots, the lesions chiefly affect the lumbar and sacral ones, and are associated with signs of meningitis; in the cervical and lumbar roots is an abnormal number, relatively, of fine slender fibres.

Simple atrophy, chiefly, affects the nerve-roots; degenerative neuritis the peripheral nerves.

The course of the lesions consists in a degeneration and absorption of the myeline-sheaths; a consecutive congestion and exudation in some cases, "the exudative or myxomatous form of the lesion," as he calls it; and in other cases, and as a third stage, this, *plus* a secondary inflammatory process—"the inflammatory form." Thus the change in nervous elements taken here to be primary, is elsewhere in the article described as secondary.

Pathogenetically, in the cord is a double morbid process:—

(1) Foci of myelitis, developing there in many and different points.

(2) Dystrophy of nervous elements (especially lateral columns), dependent upon cerebral lesions and influences.

On the spinal *symptoms* there is nothing new or that needs mention.

To some extent, the same, or analogous, remarks just made on the paper of Dr. Marie apply here also.

Cases have recently been reported from the clinic of Prof. Joffroy, of Paris, exemplifying "the spinal forms of general paralysis," *i.e.*, in which spinal symptoms or diseases precede overt, recognised cerebral symptoms of general paralysis of the insane. (*Rev. de Med.*, 1894).

In one case the chief symptoms, as of a true tabes dorsalis, had been manifest two years previously to the oncoming of cerebral symptoms of general paralysis, and the latter was associated with a very manifest improvement in the gait. At the necropsy, were found the cerebral lesions of general paralysis. The spinal cord was scarcely changed, macroscopically, but, on microscopical examination revealed (not the localisation as in true tabes, but) a diffuse myelitis of posterior and lateral columns. Therefore, the case was not one of tabes associated with general paralysis, but one of general paralysis commencing at the spinal cord and with tabetiform symptoms.

Two other cases without necropsy are taken as having the same significance.

M. Joffroy states that the association of true tabes and general paralysis is more rare than "extremely rare." As to this, I may say that about one per cent. of the cases of general paralysis are preceded by tabes dorsalis.

He also briefly mentions a case of progressive muscular atrophy preceding general paralysis by several years (the so-called amyotrophic spinal form of general paralysis). Like others, he very properly separates this from the *late* muscular wasting, so frequent in general paralysis.

Another case is mentioned, in which occurred an arthropathy of the knee, which he terms rheumatic, but which was, apparently, tabic or taboid in nature. Three years afterwards, were symptoms of the onset of general paralysis.

Besides these cases of so-called spinal forms of general paralysis simulating tabes, progressive muscular atrophy, and "rheumatic phenomena," are mentioned those in which the

simulation is of spastic spinal paralysis, and possibly, of cerebro-spinal insular sclerosis.

M. Joffroy has, therefore, seen, or known of, an interesting set of cases belonging to what some call the spinal forms of general paralysis; really meaning thereby the part affected by its incidence at first. These cases are welcome illustrations of facts already established as regards general paralysis; and, although there is nothing new in them, they offer three points as to which they present accentuation of certain features observed in other previously reported cases:—

(1) That (excepting the amyotrophic) he appears to have observed a somewhat unusual frequency and degree of improvement in the spinal symptoms, of such cases, when the cerebral of general paralysis appear. But the observation of such improvement is an old one; examples of the kind having been published more than thirty years ago.

(2) That in the case with necropsy, the simulation of antecedent tabes was more marked than it usually is in instances of the same kind.

(3) That the knee-arthopathy of a case mentioned was more than usually early in appearance (if really connected with the general paralysis at all, which, owing to the brevity of the report, is a quite uncertain point).

C.—The Ocular Disorders in General Paralysis.[1]

Of these, the chief disorder is gradual and progressive internal ophthalmoplegia; paralysis of the constrictor of the pupil and of the ciliary muscle, the former contracting to light and in efforts of accommodation; the latter acting only in accommodation and adapting the eye to distances. Hence a triple research is necessary, by the usual methods; namely, to ascertain the condition of the reaction of iris to light; secondly, the reaction of the iris and pupillary change in accommodation; and, thirdly, the power of ciliary muscle in visual accommodation. As M. Gilbert-Ballet mentions, his general results confirm the conclusions on this subject long ago published by the present writer, which were as follows[2]:—

"Speaking in general terms, I think the course of events is usually this. The pupils become somewhat sluggish to light, this

[1] M. Gilbert-Ballet. "Les Troubles oculaires dans la Paralysie générale progressive." *Le Progrès Médical*, June 10, 1893.

[2] "General Paralysis of the Insane," second edition, p. 109.

increases and some failure of their movement in accommodation often supervenes, the tendency being for the one failure to follow the other at a considerable interval. The latter failure, however, may not follow; and with immobility to light, but normal activity in accommodation, reflex iridoplegia, pure and simple, is present; or, as one might say, iridal reflex monoplegia, failure of the contraction of the sphincter iridis under the influence of light or stimulation of optic nerve. If and when, to this reflex iridoplegia there is added cycloplegia, or failure of ciliary muscle in accommodation, internal ophthalmoplegia is present. With [this] cycloplegia usually comes failure of the contraction of sphincter iridis during accommodation"; which, indeed, is an integral part of a complete internal ophthalmoplegia.

M. Gilbert-Ballet has worked out this ocular failure in greater detail, on the same lines. For one eye, considered separately, he has arranged four degrees of it: (1) paresis of movements of reaction to light; (2) abolition of these movements; (3) abolition of them, and paresis of accommodation; (4) complete paralysis both to light and in accommodation.

For the two eyes, considered conjointly, five degrees are similarly arranged: (1) loss of reaction to light only; accommodation normal; (2) both of above forms of iridal reaction weakened; (3) no reaction to light and accommodation in the eye with the more dilated pupil; simple reflex iridoplegia in the other eye; (4) same as "(3)" with weak reaction of second eye to light and accommodation; (5) complete bilateral internal ophthalmoplegia.

These ocular failures sometimes considerably precede the other manifestations of general paralysis. The nerve fibres, to iris and ciliary muscle concerned in these symptoms, come immediately from the common motor oculi nerve, which also supplies the external ocular muscles, which usually are unaffected in general paralysis. By a process of exclusion, it is concluded that the lesion, on which these symptoms depend, affects the particular nuclei of origin of third cranial nerves from which the nerve-fibres in question arise; and that it is the nuclei composing the upper group of nuclei (of third nerves), namely those whose site is the wall of the third ventricle, which—either directly or through contiguity—are mainly thus affected by the morbid process of general paralysis; whereas the other nuclei in the inferior part of the Sylvian aqueduct ordinarily escape. And when paralysis of external ocular muscles does occur in general paralysis, it is usually in those cases which are preceded by tabes; and frequently it is transitory. Nystagmus is occasionally observed;

and so is atrophy of optic disc, but, like Dr. Gowers, M. Ballet makes light of the ophthalmoscopic changes in general paralysis, which, as a rule, he finds to be slight or absent.

D.—Juvenile General Paralysis.[1]

Dr. Toulouse has collected sixteen cases of juvenile general paralysis, published by others. Fifteen years was the average age of the patients at the onset of the disease. Males were affected twice as often as females;[2] and the majority belonged to families of the labouring class.

With regard to morbid hereditary antecedents; of the fourteen cases where these were known—and counting each of them as often as it was found either with different, separate patients, or with the same patient—there was an ancestral history in six instances, of alcoholism; in four, of syphilis; in three, of mental disorder; in two, of general paralysis; and in one, of "congestive" disease.

Acquired syphilis and cranial injury have been indicated by other observers. And there were some indications of congenital syphilis in a case observed by myself.

From one aspect, juvenile general paralysis may be viewed as a precocious dementia; as the premature senility and death of a brain still young. Considered as a retrogression of the nervous elements of the brain, it is a defect of biological evolution attributable to the operation of the factors already mentioned, and perhaps to the disturbances and deep modifications stamped on the organism at, and by, puberty.

In juvenile general paralysis, at least some physical symptoms are apt to be relatively more marked than usual, more intense, and abrupt in onset; so that the youthful patients soon, perhaps, evince grave disturbances of gait, severe headache, convulsive and comatose attacks followed by paresis; also paralyses or contractures, grave emaciation, arrest of sexual development.

On the mental side, are predominantly intellectual enfeeblement and dementia; the dramatic deliria, so often observed in adult general paralysis, rarely occurring in pubescent or adolescent cases, or being slight if present.

Arrest and remission are less frequent than in the adult forms, but the course of the disease is in some instances slow.

[1] Dr. É. Toulouse, *Gazette des Hôpitaux*, August 24, 1893.

[2] This conclusion is drawn from twelve cases; it is not stated whence these were selected; and it is the reverse of that drawn by other observers.—W. J. M.

The lesions found are much the same as in the adult; atrophy being, possibly, a more striking feature.

The differential diagnosis must be carefully made from other somewhat like conditions occurring in youth. Anti-syphilitic treatment fails; in preventive management lies the only hope.[1]

E.—The Temperature in General Paralysis.[2]

The writers of the article next to be considered, conclude that in general paralysis the average bodily temperature, and the diurnal oscillations of temperature, correspond to physiological normes; that the same, practically, is true of the asymmetrical axillary differences; and that when unusual variations of temperature occur in general paralysis, their cause must be sought for in conditions not related to the pathological phenomena of that disease.

In reference to their conclusions, and to their criticism of preceding observers, one may notice that some of their cases are unsuitable, or are defectively suitable, for the purpose required. Moreover, they have made no observations on the variations of temperature taking place in connection with the paralytic and convulsive seizures in general paralysis; and yet these are the very conditions in which some of the most important and interesting variations of temperature occur in general paralysis. Besides the variations in connection with these seizures, I also often found alterations of temperature in connection with attacks of mental excitement in general paralysis; important changes and inversions of the temperature-curve in the twenty-four hours; in some conditions, subnormal temperatures; and that the temperature in general paralysis not seldom shows a preternatural mobility, a mercurial facility of change as the result of comparatively slight causes.

F.—Tumour-like Congenital Spinal Deformities in General Paralysis.[3]

A case of general paralysis is described by Dr. Bernhard Feist, in which, besides spinal cord changes resulting from the morbid process of that disease, there were several tumour-deformities of the spinal cord; partly at least, apparently of

[1] Since the above was written, a contribution to this subject has been made in the *Journal of Mental Science*, January, 1894, by Dr. Middlemass.

[2] Dr. Fr. Peterson and Dr. C. H. Longdon, *Journal of Nervous and Mental Disease*, November, 1893.

[3] *Archiv. f. Pathol. Anat.* (Virchow's), Bd. 130, Hft. 3, p. 444.

congenital origin. A number of plates accompany the record of the case, and clearly show the resulting deformity, displacement, or disjunction and separation, of several parts of the cord.

G.—The Visceral Complications of General Paralysis.

Just as this article is going to the press, I see one by M. G. Durante on the above subject. He asserts that the visceral complications of general paralysis have been but little studied, and almost passed over in silence, even in the recent monographs on general paralysis. Had he done me the honour of consulting my monograph thereon he would have read at pages 294 to 299, second edition, 1886, a very minute record, expressed in percentages, of the various visceral lesions found in general paralysis in necropsies made by myself, and much larger in number than those he cites from M. Klippel's contribution dated six years later, *i.e.*, in 1892, and twelve years later than the fairly full and exact record of those lesions in the first edition of my work, 1880.

M. Durante's contribution is chiefly an exposition of the observations and conclusions of Dr. Klippel, who has carried the study of the visceral complications in general paralysis a stage further, and who has endeavoured to establish certain points, the two chief ones of which I will briefly mention.

One is, that among the tubercular lesions found in general paralytics there is a variety which has ætiological importance; that, in fact, some cases of general paralysis are due to the late or tardy, post-infectious sclerosis—occurring remotely and without tubercular nodules *in loco*—and induced by the chronic intoxication (of microbic origin), accompanying the tuberculosis, which last, contrary to the usual rule, has in these cases reached a fourth stage. This chronic intoxication probably exercising an elective affinity on certain tissues (here cerebro-spinal), determined by a hereditary or acquired predisposition.

In such cases the pulmonary tubercular disease is slow, chronic, often latent, fibrous. At the necropsy are old fibrous or calcified tubercles, or tubercles most of them fibrous; and sometimes old cavities with hard, dry, thick fibrous walls—old lesions which for a long while had been latent. As recent changes in the lungs, may also be tubercular cavities, disseminated tubercular granulations, or caseous pneumonia with rapid softening.

My statistics (*loc. cit.*) show tubercles, caseous masses, or cavities, in 26 *per cent.* of general paralytics; traces of former cured tubercle in 12 *per cent.* If I recollect aright, without

re-examining the records, the preceding would include some of the cases with lung induration or cirrhosis, found in 14 *per cent.* (4 *per cent.*, out of this 14 *per cent.*, presenting very marked cicatrices). Consequently it is at least clear that a considerable number of general paralytics have had chronic, slow, or else regressive, pulmonary tuberculosis, before they became general paralytic.

The other chief point is that the set of visceral changes *directly* dependent upon the lesions of general paralysis in the nervous system, especially in the cortex cerebri, is "vaso-paralytic"; and amounts to a chronic congestion due to chronic paralytic dilatation of the capillaries—a constant condition but undergoing attacks of transitory increase or exacerbation, in congestive strokes. This capillary dilatation and congestion tend to form punctiform hæmorrhagic foci by vascular rupture or diapedesis. Blood pigment deposits show the chronicity of the condition.

To this process these writers attribute the visceral alterations, specially and directly dependent upon the lesions of the (cerebro-spinal) nervous centres in general paralysis; as, *e.g.*, in the lung, chronic congestion in different degrees, including hæmorrhage, broncho-pneumonia in foci, and even the apyretic pseudo-lobar varieties thereof. Thus they employ the term congestion with an unusually wide and inclusive meaning, and two-thirds of Klippel's necropsies show congestion used in this over-wide sense. Sir J. C. Brown's statistics gave congestion in 49 per cent.

My published statistics (*loc. cit.*) showed 64 per cent. with very marked pulmonary congestion; *i.e.*, in which it was the dominant condition, or else existed highly-marked together with other lesions. But besides these, it was distinctly present in many other cases, associated with more important changes of which it was, at the moment, taken to be merely an incidental accompaniment or appanage; so that, altogether, there would probably be congestion of some sort and degree in 80 or 90 per cent., or more. Nevertheless, the above writers appear to me to ascribe a somewhat too great frequency and importance to pulmonary congestion as a *direct* symptom of general paralysis; and closer examination will show that in many cases the congestion has not that meaning, and is only an indirect result or symptom, and even sometimes an incident of little importance.

In relation to the subjects of this last section, see my work, *op. cit.*, Chapters viii. and ix., which deal with the vaso-motor and trophic disorders and defects in general paralysis; also pp. 335, 194, 190 *et seq.*, 294 *et seq.*, 270-5, 341, 352-3.—W. J. M.

H.—Arthropathy in General Paralysis.[1]

In correcting "proof" of the preceding article, I take the opportunity of adding a few lines on papers which have just come to hand.

A case of arthropathy in general paralysis, with necropsy, is recorded by Dr. Lloyd. The patient, a male, aged 38, had suffered for some years with pains in the legs, which had been called rheumatic; otherwise he was apparently healthy up to one year and a-half before admission, when he had a swelling of his knees. At this time no mental symptoms were observed. Six months before admission he began to have mental symptoms. He had delusions of persecution; he became silly and weak; had a remission; then a relapse. Drawling speech began three months before his admission. After admission were a maniacal episode, and later on a grandiose delusional state. The physical symptoms partook partly of general paralytic type, partly of tabetiform type, and partly of that of insular sclerosis. The knee-joints were enormously swollen; and apparently were in the ordinary state of tabetic arthropathy (Charcot's), and presented changes of both the atrophic and hypertrophic kind. Cystitis and pyelitis preceded death.

The adhesions and chronic meningitis chiefly affected the base of the brain and the frontal lobes.

"The cord presents the type of degeneration seen usually in forms of cerebro-spinal sclerosis . . . . it does not present a typical posterior sclerosis. It rather combines two system lesions: (1) partial posterior sclerosis; and (2) sclerosis of the motor tracts from one hemisphere [the left.]

"[Dorsal cord.] (1) The posterior sclerosis is not distributed symmetrically as in locomotor ataxia, to the lesion of which disease, indeed, it presents but a partial resemblance. A large sclerosed patch occupies the anterior end of the postero-median column and the contiguous part of the postero-external column of the left side, and, less extensively, the anterior part of the median column of the right side. The postero-external column of the right side escapes almost completely . . . . The posterior root-zones practically escape (?

"(2) The motor tracts from the left hemisphere alone are involved, and these not typically. The sclerosed area in the right lateral column is not exactly co-extensive with the crossed pyramidal tract, but is more extensive. At its periphery a line of less

[1] Dr. James H. Lloyd, *Philadelphia Hospital Reports*, 1892.

degenerated fibres marks the position of the direct cerebellar tract. The region of the direct pyramidal tract on the left side presents a fan-shaped area of degeneration, sharply demarcated, and extending from the horn to the anterior median fissure. It is not co-extensive with the direct pyramidal tract." No very marked changes in multipolar cells of anterior horns in the part of dorsal cord examined; those in lumbar region not examined. Some have attributed those arthropathies to degeneration of these cells.

The above case was, at least, more or less unusual as regards the ordinary physical objective signs of general paralysis. There is not space to set this fully forth by quoting all about the physical aspects, therefore I have omitted them. But in reading it the impression comes that it was, possibly, one of insular sclerosis, or one of general paralysis *secondary to* insular sclerosis, whether or not the former arises strictly as an example of general paralysis by invasion from the vicinity; in other words, by extension by contiguity. It reminds me somewhat of some such cases published by Schultze. The above impression is not altogether set aside by the necropsy.

This subject I have dealt with elsewhere. In the above case the microscopical examination was limited to one part of the cord; and it is possible that, applied to other parts of the cerebro-spinal system, it might have revealed some scattered foci of sclerosis. And in the part of the cord so examined the change, practically, was as much, or almost so, of an insular sclerosis type as of a systemic type. Yet I fully admit the more or less "irregularly distributed disease-process," in general paralysis; indeed, I have used stronger terms in describing it, *e.g.*, "secondary scattered sclerosis." But if we accept the case as one of general paralysis, independent of insular sclerosis, there still remain the tabetiform (or tabetic) symptoms and lesions to be reckoned with. In this respect the case, however, harmonises pretty fairly with most of the few already recorded of arthropathy of the same kind in general paralysis, including two by myself (besides one not yet published).

I.—Neurasthenic Form of the Prodromic Stage in General Paralysis.

M. Gilbert-Ballet[1] records several cases of general paralysis in which neurasthenic symptoms were prominent in the prodromic stage, and sometimes for many months, or even for some years;

[1] "La période prodromique à forme neurasthénique dans la paralysie générale." *La Semaine Médicale*, 1894.

although in some cases they and the prodromic stage may be only of brief duration. The chief symptoms are headache, vertigo, obscurations, acute pain, dyspeptic disorders, hypochondriasis, general feeling of lassitude and of weakness. These, and the various other symptoms in the above cases, have all been very fully described before, as occurring sometimes in the prodromic stage of general paralysis.

The characters relied upon to distinguish these symptoms in the prodromic stage of general paralysis from simple neurasthenia are:—

In the former:—

(1) The absence of the stigmata of neurasthenia (helmet-sensation, back-ache, sacral patch, &c).

(2) The neuralgic pains; multiple, essentially mobile, and varying day by day (lightning pains, from spinal lesions, are excepted here).

(3) The sudden changes in the patient's state, *e.g.*, from a sad and downcast, despairing condition, to one of exuberant joy, satisfaction, forgetfulness of the past evils and sufferings, or assertion of recovery therefrom.

J.—The Morbid Anatomy and Pathology of the Neuro-Muscular Changes in General Paralysis.

In an excellent article, Dr. Alfred W. Campbell[1] communicates the results of his work on this subject. In all the cases examined he found the vagi nerves extensively and strikingly diseased, and this disease is connected with important clinical conditions. The phrenic nerves suffered also, but less.

In the mixed spinal nerves, and their peripheral terminations, the alteration appeared to be a combination of a parenchymatous degeneration, and an interstitial or adventitial inflammation.

In the anterior spinal nerve roots, and in the posterior roots, between the cord and the ganglia, was always a considerable degree of degeneration, but never extensive. "In the posterior root ganglia, no obvious degeneration of nerve-cells, beyond some hyper-pigmentation was noticed, and the posterior roots beyond the ganglia were but little diseased."

In the *muscles* were fatty degeneration, atrophy, and complete or partial disappearance of a number of muscle fibres, with

[1] *Journal of Mental Science*, April, 1894.

proliferation and increase of the nuclei of the sarcolemma and connective tissue. The number of motor end-plates was lessened, some were in process of degeneration; in some muscles was Eichhorst's "neuritis fascians."

The above-described changes he brings, very naturally, into line with the neuro-muscular changes in multiple neuritis of the primary intrinsic toxæmic group: referring to cases of general paralysis taken to be "purely idiopathic," and not due to syphilis or alcohol, with their effects upon the peripheral nerves.

These observations go to support me in what is already in print in the earlier part of this communication, and elsewhere on a toxic origin of general paralysis.

W. Julius Mickle.

Clinical Case.

A CASE OF PSEUDO-BULBAR PARALYSIS, DUE TO LESIONS IN EACH INTERNAL CAPSULE; DEGENERATION OF DIRECT AND CROSSED PYRAMIDAL TRACTS.

BY W. S. COLMAN, M.D., M.R.C.P.

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THE following case was admitted into the National Hospital, Queen Square, on December 31, 1892, under the care of Dr. Hughlings Jackson, who has kindly allowed me to publish it. For the notes of the state on admission I am indebted to Mr. Stoddart, the clinical clerk.

William S., aged 49, a porter, says he was perfectly well until a year ago, when his wife noticed that he dragged his left leg in walking. This ultimately prevented his working, but at the end of two months he had recovered sufficiently to be able to resume his occupation; but before long the weakness of the left leg returned, and he was quite helpless.

Six months ago he was noticed to be speaking hoarsely, and the difficulty of articulation has increased up to the time of admission. He never had any difficulty in finding the right word with which to express himself.

Two months ago he had difficulty in swallowing, noticed especially with liquids. The right leg at the same time became stiff and powerless like the left.

From this time an alteration in his mental condition was observed. He would laugh or cry incontrollably at the slightest thing. He has had no hallucinations.

Previous history.—He denies syphilis and its sequelæ. His wife has never been pregnant. No history of alcoholic excess. He has been subject to much physical strain in his occupation as porter.

Present State.—Patient is a well-porportioned, muscular man, complaining of weakness and stiffness of both lower limbs, inability to walk, and difficulty of articulation.

Mental Condition.—He has a vacant expression, and is provoked by the merest trifles into loud laughter, ending in an inspiratory whoop, or in unreasonable weeping. He complains of loss of memory. His mental condition generally seems dull and confused, but his difficulties of articulation are so great that investigation is extremely difficult. He does not complain of headache or vertigo. He sleeps well at night, and there is no drowsiness during the day. No delusions or hallucinations can be elicited.

Speech.—He finds great difficulty in articulating words, to such an extent that he resents being made to speak, and prefers to express himself by signs. Phonation hardly ever gets beyond a hoarse whisper in speaking. He cannot pronounce the explosive consonants, and his speech is very thick, as if his tongue was too large and almost filled up the buccal cavity. There is no true aphasia.

Motor Symptoms.—There is weakness of the muscles of face, arm and leg on both sides, more marked throughout on the right side.

Face, &c.—There is distinct weakness of both sides of the ordinary cerebral type, and there is also considerable weakening of the occipito-frontales, and orbicularis palpebrarum.

The left side acts better than the right, so that the mouth is drawn to the left in laughing, and when he attempts to shew his teeth, the left naso-labial fold is more marked than the right. He is quite unable to whistle.

Tongue.—All movements can be performed, though defectively, *e.g.*, he can only prostrate the tip of his tongue as far as the lips. There is no obvious wasting of the tongue and no fibrilliary twitching.

Palate.—Soft palate, deficient both in voluntary and reflex contraction on both sides. (It afterwards became completely paralysed.)

Larynx.—Both vocal cords congested, but move well (Dr. Semon).

Neck.—There is considerable weakness of sterno-mastoids and trapezii and probably deeper muscles, so that he cannot sit up in bed without having his head supported.

Upper Limbs.—Almost all movements of both upper limbs can be carried out, although feebly. Movements are distinctly weaker on the right side.

Trunk.—Muscles of the back are very weak. He cannot sit up, even with the aid of his arms.

Lower Limbs.—Extremely weak, and occasionally rigid, but not always so. He is quite unable to stand.

Superficial Reflexes.—Plantars increased: abdominal and epigastric not obtained.

Deep Reflexes.—Knee jerks much exaggerated. Ankle clonus present on both sides. Wrist and elbow jerks increased. Jaw jerk present.

Organic Reflexes.—Automatic emptying of bladder. For some weeks before his death he also passed his motions unconsciously.

Sensation.—Sensation to all forms of stimuli was quite normal in every part.

There were no indications of disease in any of the thoracic or abdominal viscera. Four months after admission he was attacked with general eczema. He seemed ill, and his temperature rose to 101°. In a few days there were signs of general bronchitis, and he died from this complication on May 5, 1893.

Examination of the Brain.—After hardening the brain, &c., in Müller's fluid, the following conditions were found:—

The convolutions showed a little general wasting, but were otherwise normal.

The vessels at the base of the brain were atheromatous, but elsewhere they were natural and the membranes thin.

On making vertical sections through the internal capsules, they were found to be soft and friable. Under the microscope sections showed many bundles of completely degenerated fibres separated by normal bundles, at the point where the fibres streamed out of the capsule into the crus, and anterior to this were several small areas of softening. Many of the small arteries in this neighbourhood were completely thrombosed. In the right capsule was a patch containing hæmatoidin crystals, evidently the site of a small hæmorrhage.

The degenerations could be traced in the pyramidal tract to the crus, pons and medulla, and in the whole length of cord in the lateral pyramidal tracts, and as far as the third dorsal in the direct pyramidal tract. The degenerations were more extensive on the right side of the cord than the left.

The cells of the hypo-glossal nucleus and the anterior cornual cells of the spinal cord were not diminished in number, and were quite normal in appearance.

The other organs shewed no change of importance. In a case recorded by Drs. Hughlings Jackson and Taylor, in which I

examined the cord, there were very similar symptoms and lesions in the same position, but in that case there was no degeneration of either direct pyramidal tract. I have been able to find no reason for the difference in the two cases.

Remarks.—The course of the disease in this case closely resembled one of bulbar paralysis, with descending changes in the cord. A close investigation of the history, in the light of the *post-mortem* examination, shows that there were two attacks of left hemiplegia, followed at a long interval by one on the right side, and coincident with this the development of marked bulbar symptoms, as in the cases recorded by Barlow,[1] Hughlings Jackson and Taylor,[2] and Lépine,[3] in cases of double hemiplegia. The attacks of hemiplegia were peculiar, in that they commenced with comparatively slight paralysis, which afterwards increased. This would seem to have been due to repeated small lesions (small hæmorrhages and thrombosis of small arteries) occurring at intervals in the same neighbourhood, such multiple lesions being evident on microscopic examination in each internal capsule.

In the absence of any reliable history as to the onset of the symptoms, it must always be difficult, or even impossible, to diagnose between double hemiplegia and bulbar paralysis, with paralysis of the limbs (amyotrophic lateral sclerosis). The chief points of divergence seem to be :

(1) That in double hemiplegia the affection of the limbs is not quite symmetrical, but usually distinctly more marked on one side than the other.

(2) The absence of any marked wasting of the tongue in double hemiplegia.

(3) The preservation of electrical excitability of tongue and palate in double hemiplegia, while there may be some diminution of response to both forms of current in bulbar paralysis.

[1] *British Medical Journal*, 1877, vol. ii. [2] *Lancet*, 1892, vol. ii.
[3] *Revue de Médicine*, 1893.

Reviews and Notices of Books.

Grundzüge der physiologischen Psychologie. Von WILHELM WUNDT, Professor an der Universität zu Leipzig. Vierte, umgearbeitete Auflage. Leipzig, Verlag von Wilhelm Engelmann, 1893. Vol. I., pp. xvi., 600. Vol. II., pp. xii., 684 (Index, pp. 649-684), 143 and 94 figures.

Grundriss der Psychologie, auf experimentelle Grundlage dargestellt. Von OSWALD KÜLPE, Privatdocent an der Universität, Leipzig. Leipzig, Verlag von Wilhelm Engelmann, 1893. Pp. viii., 478 (Index, 472-478). Ten figures.

WHEN the "Physiological Psychology" first appeared, in 1874, it was as a single volume, of 870 pages.[1] Lotze and Fechner had already done much to prepare the way for the "new" psychology: but it is hardly an exaggeration to say that the publication of Wundt's encyclopædic work, and the opening of his laboratory in 1879, mark the actual birth of the science. Much of the factual material of this first edition was drawn from the author's already published writings: the *Lehre von der Muskelbewegung* (1858), the *Beiträge zur Theorie der Sinneswahrnehmung* (1862), the *Vorlesungen über Menschen und Thierseele* (1863), the *Lehrbuch der Physiologie* (1865), the *Physikalischen Axiome in ihrer Beziehung zum Causalprincip* (1866), the *Handbuch der medizinischen Physik* (1867), and the *Untersuchung zin Mechanik der Nerven und Nervencentren* (1871). The rest was culled with pain and travail from the most varied sources: Johannes Müller, Fechner, Helmholtz, and a hundred others. The "formative influences" mentioned in the preface are the works of Kant, Darwin, and Herbart.[2]

[1] First part published separately, 1873.

[2] This edition was reviewed by Professor Sully in "Mind," O.S., No. I., pp. 20 ff.

The second was a two-volume edition (1880). A French translation of it (Paris, 1886) was issued just before the appearance of the third German edition. French being more generally understood than German, and Wundt's technical German being none of the easiest, this translation was and still is much used and cited. It is, of course, hopelessly antiquated: in fact, fourteen years old, despite its date of publication. The third edition[1] itself (1887) showed a great advance in the progress of the science. So far from its being now difficult to get material together, the difficulty was that of selection from and discrimination within a truly vast literature, written in all the chief languages of Europe. Not only had there arisen the new school of professional psychologists: psychological problems had attracted the attention of scientists and philosophers of every school and interest. But even more formidable than in 1887 was the task of revision in 1893. The fourth edition is, it is needless to say, larger than the third. That, however, is not all; it is very different from it. In the last six years neurology and experimental psychology have changed, as regards fact, method, and theory; and it is attempted to do justice to these changes, in the revised chapters which treat of the two disciplines in question. Moreover, Wundt's own views upon matters of systematic psychology have undergone important modifications, which would of themselves suffice to justify republication of the work, with the title of a new edition.

It must always be a matter of regret to psychologists that Lotze published no review of the "Physiologische Psychologie." Without disrespect to living representatives of the science, it may, I think, be said that he was the one man in every way qualified to undertake the task of criticism and appreciation—having had, like Wundt himself, the preliminary training of a physiologist and physician. It would in oneself be no less than impertinent to attempt a "review," in the sense in which the word has just been employed. The object of the present notice is far more restricted; it is: (1) To furnish those who are already acquainted with the third edition of the work with an outline of the alterations introduced into the text of the fourth; and (2) To give readers who come to the latter, as to an unknown book, some idea of the general contents of it.

The *Introduction*[2] has suffered but little change. In it the

[1] Reviewed by Professor Cattell in "Mind," O.S., No. LI., pp. 435 ff.

[2] 3RD ED., I., 1-20; 4TH ED., I., 1-20. *Omitted:* 3rd Ed., p. 3. *Added:* 4th Ed., pp. 8, 9, *Diejenigen. . . Expanded:* 4th Ed., pp. 4-6. *Condensed:* 3rd Ed., pp. 16 midd.—18.

author treats, as before, of the problem of physiological psychology and of the presuppositions of psychology in general. A reason is given for the retention of the adjective "physiological" in the title of the work. The expression "experimental psychology" is the equivalent of "psychology," as at present defined. "Physiological psychology" (1) pays more special regard to those psychological investigations whose instruments are physiological than to any others, and (2) discusses the physiological basis of mental life: its field, therefore, is at once narrower and wider than that of "psychology." A sharp criticism of hypnotic "experimentation" refers the reader to the recently published brochure "Hypnotismus und Suggestion."

Part I.[1] deals with the physical basis of mental life, and is mainly taken up with an exposition of the relation of the nervous system to the mind. Chapters and sections are entitled and arranged as in the third edition. The discussion is of immense value, as being the only adequate treatment of the subject extant, from the psychological point of view. Students of neurology, who come to it solely for the purposes of their own science, will probably be disappointed in what they find; it will seem to them that more space should have been devoted to the quite recent discoveries and inferences of nervous physiology. But the attitude would not be a fair one. There is ample indication that Wundt's erudition has kept pace with neurological research, and that, if his physio-psychological position has not greatly changed, this is due to a belief that there is nothing in the many lately-published monographs which calls for its radical alteration.

The chapters contained in this *Part* are six in number: (I.) on the organic development of mental functions; (II.) on the structural elements of the nervous system; (III.) on the gross-anatomical development of the nerve centres; (IV.) on the course of neural conduction paths; (V.) on the physiological functions of the central parts; and (VI.) on the physiological mechanics of nervous substance. Some alterations have naturally been intro-

[1] 3rd Ed., I., 21-288; 4th Ed., I., 21-280. *Omitted:* 3rd. Ed., pp. 64, last sent. of § 1; 80 note; 87 last sent. of midd. §; 115 last sent. of §; 115 *Ebenso* to end of small pt.; 162 last sent.; 198 *Da nun alle* to 199 end of §; 215 large pt.; 217 second §; 250 last half of last sent. of § 1; 266 small pt. to 269 top; figg. on pp. 30, 72, 74, 75, 84, 86, 93. *Added:* 4th Ed., pp. 34 top to midd.; 91 small pt.; 98 last sent.; 138, 9 *Wahrscheinlich . . . . .*; 166 last sent.; 244 two sents. in middle §; 274 *Aus diesen . . . . .*; 278 last sent. of § 1; figures on 34, 130. *Expanded:* 4th Ed., pp. 38, 9; 127; 130; 192; 201 note; 211 note; 214 second §; *Condensed:* 3rd Ed., pp. 36; 66, 7; 68; 91 note; 111 last part; 166 end of small pt.; 194 last half; 211-3; 231-4. *Modified:* 4th Ed., pp. 81, end of § 2; 146; 188 last sent. of § 1; 228 end; 230, 1; figg. on pp. 122, 141.

duced in the second of these;[1] "collaterals" were not thought of in 1887. Indeed, much of the minute work of Ramón y Cajal, Kölliker, Retzius, Golgi; of the gross work of Edinger; and of the functional investigation of Mosso and Horsley is quite recent.[2] In the fourth, the section on the fillet (laqueus) has been re-written; the twofold nature of the optic and acoustic nerves (as at once centripetal and centrifugal) is discussed; and the probable indifference of function of commissural fibres in the lower central parts remarked. Portions of the section on paths of conduction to the cerebral cortex have been remodelled, though its general conclusion remains as before. The schematic figures of the lines of conduction through *pons* and *cerebellum*, and in *pons*, *cerebellum*, and *cerebrum*, have been brought up to date: but, unfortunately, at the same time reduced in size. The section on sleep (chapter v.; automatic functions: *cf.* chapter xix.) has been revised. Recent experiments on the total extirpation of both hemispheres, in dogs and birds, have led to modification of the section on cerebral function. In the theory of central innervation (chapter vi.), more stress is laid than was previously the case upon the nutritive influence of the central substance on the fibres connected with it. Finally, the schematic and hypothetical representation of the apperception-organ has been changed for the better, and the textual consideration of it to some extent altered. The whole discussion remains modestly in the small print, to which it was relegated in the third edition. One could have pardoned the author, if persistent misunderstanding of his theory had led him to restore it to its original type.

Part II.[3] is concerned with sensation. The chapters are

[1] The phrase "unipolar cell," despite Golgi's "monopolar" is surely a survival from the older neurology.

[2] Lenhossek, Van Gehuchten, Obersteiner and Cunningham do not appear to be mentioned.

[3] 3rd Ed., I., 289-544; 4th Ed., I., 281-600. *Added:* 4th Ed., pp. 286 top; 293 last sent. of § 1; 295 last sent. of § 1; 303 *Für die Temperaturreize* . . .; 310 last sent. of § 1; 323 *In der That* . . .; 333 after italics in § 3; 356 ff.; 361-5; 389 ff.; 394; 396 last sent. of § 2; 417 last § of large pt.; 455 midd. §; 462 to sents. at top; 464 *Stosstöne*; 494 top sent.; 509 *Uebrigens* . . .; 514, 5 *Die günstigen* . . .; 517 *Alle diese* . . .; 520 last word to end of § on 521; 529 midd. §; 542-4; 549 §; 550 midd. §; 558 § 2 to 563 top, much added; 582-6; 596 to end; figg. on 300, 308, 315, 361, 362, 363, 373, 374, 409, 459, 502, 506, 542, 543. *Omitted:* 3rd Ed., pp. 328, *Da aber* . . .; 334 last sent. of § 1; 348 *Dabei ergänzen* . . .; 349 § 2; 359 last sent. of § 1; 441 last sent. of § 1; 578 last sent. of small pt.; 530 top 6 lines; figg. on 300, 305, 315, 324, 327. *Expanded:* 4th Ed., pp. 297, last sent. of § 1; 306, 7 *Von den* . . .; 314, 5; 319, 20 *Trotz* . . .; 327 top; 328, 9; 329 to midd. of 331; 342-58; 359, 60 sent.; 360, 1; 372 ff.; 381 ff.; 383 ff.; 387 ff.; 396 last sent.; 404, 5; 410 to 414 top; 417-9; 419-22 top; 422 midd.; 422-6; 426-33; 440 1; 450, 1; 464, 5, 6; 466; 468, 9; 472, 3; 473-81; 500-3; 505, 6; 515, 6; 521 midd. §;

headed as in the previous edition: (VII.) the origin and general properties of sensation; (VIII.) the intensity, and (IX.) quality of sensation; and (X.) the affective tone of sensation. But this is one of the two *Parts* which at first sight show the traces of revision most clearly. Indeed, Wundt tells us in the *Preface* that such an impression was intended. Experimental psychology is to-day an independent science; it has its own methods and its own instruments,—methods and instruments as peculiar to it as are those of chemistry to that discipline. It seemed well, therefore, to incorporate a somewhat detailed account of these in the "Physiological Psychology." And, since sensation has proved to be one of the most fruitful departments of experimental psychological research,[1] we find technique and procedure discussed here far more exhaustively than in the book of 1887.

I have said that Wundt is the father of experimental psychology. The younger generation of laboratory-trained psychologists is divided, as things are, into two groups: an orthodox school, which accepts Wundt's cardinal doctrine—that sensation is not the sole elementary mental process, but that conation is as radical as sensation chronologically, and prior to it logically; and a school of dissent, which denies primitiveness to any other constituent of mind, explains affection (pleasure-pain) and conation in terms of sensation, and seeks to affiliate experimental results to the theories of the older English associationist psychology. Both parties point to experience as their witness. The former says: We find in consciousness we experience, a conative process, which is definite as regards quality, intensity, and duration. The latter: We find only sensation-processes; an idea of

536; 552 3 last sents. *Condensed:* 3rd Ed., pp. 291, 2; 321, 2; 361; 365; 380, 1; 388, 9; 395; 421 midd. §; 426 *Zugleich . . .*; 432 § 2; 433; 470, 1; 490; 499, 500; 503; 513 to top of 515; 543, 4. *Modified:* 4th Ed., pp. 281; 282; 287; 299 last §; 327 § 2; 366, 7; 370 fig.; 370, 1; 438-40; 448 top; 468 end of § 1; 488; 500 end of § 1; 523 midd. §; 526 end of § 1; 529 top.

[1] I should like to take this opportunity of protesting against the widely spread and frequently expressed view, that experimental psychology can never deal with the "higher" mental processes. "Experimental psychology," as Wundt puts it, is just "psychology," the impossibility of experiment is as wide as mind. To answer the objection more concretely: (1) the sensation is the easiest psychological material to work on, and therefore has, naturally, been taken first; (2) the doctrine of sensation is by no manner of means completed so that, in assigning subjects of investigation to comparatively unskilled students, directors of laboratories tend to choose them from within its limits; (3) the statement is disproved by facts; fusions of such a high order as attention, emotion, and apperceptive ideational combination having been experimentally investigated under various aspects. Experimental psychology simply needs time and trained workers. Any restriction of the phrase such as that which the word "psycho-physics" has undergone is to be deprecated; *cf.* the remark made at the conclusion of the notice of Dr. Külpe's *Grundriss*, below.

movement is to us the will to move, strain-sensation complexes are attention. And experience must be the final appeal: it is only a matter of better or worse introspective analysis. No one recognises this more explicitly than Wundt himself, whose theory of conation or apperception is a strictly empirical, experiential theory—as "unmetaphysical" as most current hypotheses in the natural sciences are "metaphysical." Sensations, therefore, are defined, at the outset of this *Part*, not as the simplest elements of consciousness, but as the simplest elements of ideas: the idea being further described in Vol. II., as the mental image of an object or process of the external world.

There is but little alteration in chapter vii. A new paragraph deals with the terminal apparatus of the temperature sense. The treatment of the ear is modified to correspond with alterations in the treatment of the auditory idea in *Part III.*; that of the eye to correspond with the altered *opticus* schema in *Part I.*—a schema, by the way, which is here turned to account for the explanation of the "projection" of hallucinations and fancy-images. The critical and historical discussion of the doctrine of specific energies is much fuller than in the previous edition.

The detailed account of the psycho-physical measurement-methods (chapter ix.), on the other hand, is greatly enlarged. These methods are, of course, employed with the object of obtaining exact knowledge about mental processes, considered under various aspects. But their elaboration and formulation have proved to be so difficult, that there has arisen a very considerable literature, devoted to the interests (mathematical and other) of the methods themselves, without regard to the conscious content for whose examination they were primarily designed. This literature is not psychology, but it is useful to the psychologist: and, so far as that is the case, it is reproduced in Wundt's summary. Much new matter is also introduced in the discussion of Weber's law in the various sense-departments. The summing-up upon the general validity of the law, is longer and more cautiously worded than it was six years ago; but the general verdict is still favourable, despite the complication by proof of the existence, under certain conditions, of a direct proportionality between stimulus and sensation.[1] Similar alterations are noticeable in chapter x. The cutaneous and deeper-lying sensibilities are in particular subjected to a more exhaustive qualitative analysis. "Sensations of inner-

[1] It is curious that the degrees on the brightness scale (black through grey to white) are treated throughout as though they indicated intensity-differences only, and not quality-differences. See, however, p. 505.

vation" and the "sense of feeling" are finally given up, on account of the liability of the expressions to cause misunderstanding. The exposition of Wundt's own theory of light-sensations, which is the only non-component hypothesis in the field, is very opportunely lengthened and clarified.

The author's attitude to pleasure-pain content remains more conservative than the treatment of it in the second edition of the *Vorlesungen* might have led one to expect. The younger Wundtians are apt to look upon sensation, affection, and conation as equally primitive and equally elemental independent processes.[1] Such a view is not inconsistent with Wundt's theory of affection—that it is the mode of reaction of conation upon sensation; but he prefers himself to speak of the "affective tone of sensation," and to look for the conditions of pleasure or pain in the sensation, rather than the stimulus-process. I cannot here enter upon the reasons which lead many psychologists to a severance of affection from sensation, and to the preference of its correlation with conation. They seem to me to be weighty, and to be implicit—indeed, partly explicit—in Wundt's own system. Chapter x. contains much that is new: in the analysis of the concrete pain-feeling, the added section on the physical correlates of the sense-feelings, the discussion of theories of the origin of these.

Part III.[2] on the formation of ideas, contains four chapters: (XI.) general considerations; ideas of touch, movement, (XII.) ambition, and (XIII.) vision; (XIV.) the elementary æsthetic feelings. The tactual idea and the theory of tactual space are handled at greater length than before, and their mode of treatment modified, just as was the case with the cutaneous and subcutaneous sensations in Vol. I. The phrase "psychic synthesis" (as descriptive of the sum total of mental processes underlying the construction of non-visual space) has been replaced by the less ambiguous "extensive fusion." "Fusion," to Wundt, is still no more than very intimate association. He

[1] Independent, of course, only in abstraction; not in the concrete mentality of the individual.

[2] 4th Ed., II., 1-254; 3rd Ed., II., 1-224. *Added:* 4th Ed., pp. 1 *Beide* . . .; 13 *Dies erklärt* . . .; 19 last sent.; 21 *Hierbei* . . .; 28, 9; 33 *Werden dagegen* . . .; 71 top §; 71-4; 87-9; 95, 6; 100 last sent. of § 1; 122, 3; 129 *Die geschilderten* . . .; 137 midd. §; 142 midd. §; 155, 6; 172, 3 sent.; 245-8; figg. on 123, 129, 146 ff., 201, 241. *Omitted:* 3rd Ed., pp. 3 *Nun haben* . . .; 45 top sent.; 72 3 sent.; 80 last § of small pt.; 198 top sent.; 207 *Ebenso* . . . *Expanded:* 4th Ed., pp. 27, 8; 29-32; 50, 1; 60; 66, last §; 94, 5; 133; 139, 40; 146-52; 240, 1. *Condensed:* 3rd Ed., pp. 11, 12; 21-3; 24 *Wenn man* . . .; 54 second §; 80 1st two §§; 115 midd. §. *Modified:* 4th Ed., p. 23; 26; 38; 41; 83; 101, 2; 140-2; 142, 3.

does not separate fusion (= intensive or qualitative combustion of elements, in which the constituents are overshadowed by the resulting total-process) from association (= temporal or spatial combination, when the constituents are in the result as clear as or clearer than they were in isolation), as, *e.g.*, Külpe does. This is shown in the added pages on tonal fusion in chapter xii.; the other more important alterations in which are the fuller discussion of auditory localisation, and the introduction of new sections on the temporal association of auditory ideas. The next chapter shows the traces of revision most clearly in relation to optical illusions. It is, of course, one of the most thoroughly Wundtian parts of the work. Of the three moderns mentioned by Professor Sully[1] in 1876, as mainly responsible for the "derivative" or "empiristic" theory of psychological space, Helmholtz vitiated much of his reasoning by confusing logic with psychology, while Lotze adopted a nativistic metaphysic, which deprived his "local organs" of their real importance. Wundt's views have been subjected for a quarter of a century to the tests both of internal and external criticism; and are, in their present form, typical of the opinions of one of the two main schools of space-psychology. Chapter xiv. contains new paragraphs, dealing with the relation of the elementary æsthetic feelings to other forms of the feeling-process.

Part IV.,[2] of consciousness and the train of ideas, is the other of the two *Parts* which show the most obvious and unmistakable evidence of revision in the new edition. Its chapters treat (xiv.) of consciousness; (xvi.) of apperception and the train of ideas; (xvii.) of ideational combinations; (xviii.) of emotional phenomena; and (xix.) of disturbances of consciousness. To be noted at once are certain alterations in systematic psychology. In the first place, the process of apperception is more clearly separated out from its conditions and associated processes; those determinants of a train of ideas which are to be looked for in the general psycho-physical trend

[1] "Mind," O. S., I., p. 24.

[2] 3rd Ed., II., 225-462; 4th Ed., II., 255-559. *Added:* 4th Ed., pp. 276, 7; 291, 2; 294 last sent.; 311 note; 315-7; 318 second table and remarks to 319 top; 346 *Martius*; 357, 8; 379, 80; 429-31; 436; 441-447; 480 *In der Ausbildung* . . .; 480-2; 497-501; 508 *Wird ein Streben* . . .; 546 *In der Neigung* . . .; figg. on 291, 359, etc. *Omitted:* 3rd Ed., pp. 253 last sent. of § 1; 271 *Zu einem grossen* . . .; 291 last sent. of long §; 410 two last sents. of top §; fig. on 297. *Expanded:* 4th Ed., pp. 266-8; 271-5; 275, 6; 279-82; 282-6; 295-300; 322-44; 344, 5; 347, 8; 360-2; 363 long §; 420-9; 431 § 1; 457-64; 466-75; 543; 545, 6; 547 midd. §; 552-4. *Condensed:* 3rd Ed., pp. 240-2; 244, 5; 285, 6; 409 midd. §; 438-40; 453, 4; 456-9. *Modified:* 4th Ed., pp. 266; 300, 1; 348, 9; 370 midd.; 408; 408-20; 431.

or disposition of an individual at a given time, being now definitely marked off from the conative elements in the whole mental content of that time. The attention is regarded, in essential, as a phenomenon of inhibition (chapters xv., xvii). Secondly, time-ideas (*cf.* chapter xii.) are discussed in a new section, principally upon the basis of Meumann's recent investigations; extant theories of the "time-sense" are criticised; and the form which an adequate theory must take is pointed out. The psychology of time is not, of course, established upon so secure a basis as that of space; but light, factual and methodological, is beginning to break in upon us. I hardly know of any series of special researches, the following out of which shows more distinctly the difficulties and special fallacies that beset the path of the psychological experimenter, than that dealing with the "time-sense," which begins with Vierordt and Mach, and ends with Schumann and Meumann. Thirdly, the doctrine of association of ideas has been very thoroughly revised; a long section added, on the theory of association; and, as indicated above, the apperceptive factors sharply distinguished from the associative. An adequate discussion of any one of these three changes would in itself demand several printed pages. Here they can only be barely mentioned, with the remark that they are all in the direction of true psychological progress, and that there is a complete absence of indication of that fossilisation of thought, which maturity is so apt to bring to speculative and constructive minds. No one who has had the privilege of personal intercourse with Wundt can have failed to be impressed by his openness to new ideas.

Attention may be called to some few of the other alterations of *Part IV.* In the first place, the technique of mental chronometry—a department of investigation in which Wundt's first experiments date back to 1860—and of the "time-sense" experimentation, is elaborately described; figures of apparatus given; and typical sources of error pointed out. The recent confirmations of the view that the so-called "fluctuations of attention" are really what their name states them to be, and the present state of the controversy, as regards the two fundamental forms of the simple reaction, are set forth fully and clearly. The experimental basis of association-theories is seen to be still very inadequate, though the most is made of the little work that has been done. An entire section is added in chapter xviii., on the general interconnexion of affective processes. For most of these changes, readers of the *Philosophische Studien* will have been to some extent prepared.

Part V.[1]—on will and the external voluntary action—has remained practically untouched. It contains chapters (xx.) on the will; (xxi.) on the influence of the will upon bodily movement (automatic and reflex; impulsive and voluntary); and (xxii.) on expression of emotion. I have already said that the doctrine of conation is the hinge upon which the whole of the Wundtian psychology turns. For a complete understanding of this doctrine, the present chapters should be read in connection with parts of the *Ethik* and *System der Philosophie*, with certain of the *Essays*, and with various articles in the *Phil. Studien*. The titular limitation of the work under consideration would of itself indicate the necessity of such comparative reading.[2]

And it has, naturally, been found equally unnecessary to change the exposition of *Part VI.*,[3] which treats in two chapters—(xxiii.) metaphysical hypotheses as to the nature of mind; (xxiv.) general outlines of a theory of our inner experience—of the origin of mutual development.

If Wundt has spared no pains to make "physiological psychology" complete, within its limits, he has himself called our attention to the fact that these limits are not so wide as those of the science of "psychology." Indeed, as I have pointed out above, this would have been a necessary inference from the scheme and plan of the work. After a brief introduction to the general subject, we are shown in detail the bodily structures and functions with which mentality is correlated. We then enter upon the study of the most objective of simple mental processes—sensation; which is handled in both general and special physiological reference. In the third place, we examine the ideas of sense, so far as this is possible under the assumption that they are simply groups or complexes of sensations, *i.e.*, omitting for the time being the consideration of the conative factor in their composition. Up to this point the task of correlation has been, in theory at least, fairly easy. But now our faculty of analysis is to be more severely tasked. We are taken to the concrete mind; its "states" of attention and

[1] 3rd Ed., II., 463-530; 4th Ed., II., 560-625. *Omitted:* 3rd Ed., pp. 480 last. sent of § 1; 480 *Eben darum . . .*; 482 last sent. of midd. §; 486 *Von psych. Seite . . .*; 489 last part of §; *Condensed:* 3rd Ed., pp. 483, 4. *Modified:* 4th Ed., pp. 575 ff., title.

[2] In the same way we shall go to the *Logik*, in order to supplement the psychological doctrine of the "Phys. Psych." on other sides.

[3] 3rd. Ed., II., 531-554; 4th Ed., II., 626-948. *Omitted*: 3rd., Ed., p. 544, last sent. of § 1. These footnotes will have shown what it is hardly needful to mention, that a very large number of textual modifications has been entirely passed over in the above notice. Some of them are of considerable psychological importance; *e.g.*, the analysis of sense-pain.

emotion, its "trains" of ideas, and the abnormal conditions into which it is liable to fall. Here the physiological processes co-ordinated with the mental are of a higher order of complication and less exactly known: care is needed, lest the investigator, anxious to make his parallel complete, fall into a barren schematism, which shall tend to hinder the later acquisition of real knowledge. Lastly, and with the greatest difficulty, we approach —always with the physiological reference—the concrete study of that side of mind which we have learned to know in its simpler form in the "states" and "trains" already handled—the will. And a final section co-ordinates the different views which we have been led to adopt as the investigation proceeded, and shows us the justification of the assumptions which we made, as working hypotheses, at the outset of our inquiry.

The programme is coherent and complete in itself: but, to quote from Wundt again, it contains both more and less than would the programme of "psychology." We require from the author another special psychology: a "social psychology," which shall deal with myth, language, and custom. The lectures on this subject, which have been delivered for several years, will surely one day appear in book form. May we not also require from him a systematic psychology,[1] freed from *any* such definite reference? An example has already been set by Professor Ladd, who publishes a "Psychology" before a second edition of his "Physiological Psychology" (1887) has been called for.

The two volumes are well printed and have good margins; the paper—*O si sic omnes!*—is a deep cream, and not white;[2] the cuts are clear, and better set than in the third edition. Dr. Meumann has furnished an excellent index of matter. Is it ungenerous to end with the wish that as good an index of names had also been provided?

Dr. Külpe's book is dedicated to Wundt, and is throughout influenced by Wundtian modes of thought, the other influence most distinctly traceable being that of G. E. Müller. It is undoubtedly the best text-book of experimental psychology extant; and—what is not necessarily implied in that statement—it is a thoroughly good text-book, regarded on its own merits. The

[1] *Cf.* J. W. "Mind," N.S., No. 9, p. 143.

[2] If one is to judge from present indications, it will still be long before publishers and public recognise the fact that there are a real right and wrong about the make of a book: its size, type, tint of paper, &c. The glaring white of so much note paper and of so many *éditions de luxe* is hopelessly "unpsychological."

value of its parts, from this point of view, is of course very different. Some chapters are based upon an extensive experimental literature; others, the experimental material for which is still scanty or untrustworthy, the author has been obliged to construct as best he could from these scanty materials, and to elaborate by the aid of hypotheses erected upon admittedly insecure foundations. But it is no small thing to have written the first adequate manual of the science. And the high reputation which Dr. Külpe had already won by his published articles will certainly be increased by the quality of the present book.

I can only note the contents and its arrangement in the briefest way. The *Introduction* (pp. 1-29) deals with the problem of psychology, its methods and the assistance which it receives from related disciplines, and with psychological classification and literature. *Part I.*, on the elements of consciousness (pp. 30-283), falls into two sections, entitled respectively "Sensations" and "Feelings." Prefixed to the detailed consideration of the properties of sensation is a chapter (1) on sensational analysis, which, in pædagogical reference, must be termed no less than masterly. The author's long experience as assistant in Wundt's Institute has given him a familiarity with the use of the psycho-physical measurement-methods, and an insight into the difficulties presented to the beginner in the study of them, which have combined to make his exposition a model of conciseness and lucidity. The following chapters treat of (2) the quality and (3) intensity of peripherally excited sensations; and, under the title "centrally excited sensations," of (4) reproduction and association. The discussion of the latter processes is more than a text-book *résumé*: it is a real and independent contribution to systematic psychology. For that very reason, however, it does not stand upon the plane of the previous chapters; its conclusions are, from the nature of the case, more individual and debatable. In the section on feeling are included paragraphs dealing with the question of an elementary conative quality.

Part II. (pp. 284-437), on the combinations of the conscious elements, falls similarly into two subsections; on "Fusion" and on "Association." I have above called attention to the difference between these two forms of combination, and think, with the writer, that they should be separately handled, and that separate theories are required for their explanation. The fusions discussed are (1) those of tone sensations, (2) of brightness with colour, &c., and (3) of sensation with feeling (impulse, emotion). Under the association title we have chapters on (1) the spatial properties and relations of sensations; (2) the temporal properties and

relations of the elements of mind; and (3) temporal and spatial association. Cases of the latter are visual contrast, and the simple reaction (= the exact type of an "action.") The "association of ideas" is not further treated of (*cf.* *Part I.*, ch. 4).

Finally, the "state" of consciousness forms the topic of *Part III.* (pp. 438-471). "States" of mind are attention, voluntary thought, self-consciousness, dreaming, hypnosis, &c.

The author professes himself a disciple of Wundt, as regards the latter's doctrine of apperception. But, ingeniously as he reconciles his own view with Wundt's terminology, and dexterously as he takes advantage of Wundt's latest modifications of his theory of attention, the fact cannot be overlooked that the standpoints of the two psychologists are at bottom different. Effort—the quality of will—is reduced to a complex of sensation qualities (p. 275). There is no special conscious content, "internal activity" (p. 440). But this is hardly a restatement of the teaching of the "*Physiological Psychology*" II., 266, ff., to say nothing of other passages in Wundt's writings. A feeling of loyalty may have prompted Dr. Külpe to minimise his divergence from his teacher: but the difference is certainly there.

I do not intend, however, to give here a detailed criticism of the work. Only one remark may be made in conclusion: that the arrangement of the book does not seem to be the best possible. To treat of emotion and impulse before attention has been touched is, I think, bad pædagogical policy, even if the order of treatment do not rest upon faulty psychological analysis. The elementary will-quality is out of place among the feelings. Expressions of the emotions should be dealt with under the heading of "association" (*cf.* p. 343). The association of ideas might well have been spared a few more pages—again for pædagogical purposes—in the chapter discussing contrast and action. That the author is upon the track of the right arrangement of an experimental psychology, I have little doubt, but I incline to think that a fear of over-schematisation has led him to depart from the logical consequences of his original plan.

Find fault as one may, however—because the contents are not ideally arranged, because the book is part manual, part constructive psychology, because it is so predominantly German in reference and appreciation—the fact remains, as stated at the outset, that Dr. Külpe has produced a thoroughly good text-book of experimental psychology. It is none the less welcome, as a complete disproof of the notion that the science has covered no other ground than that of sensation.

E. B. TITCHENER.

Métaphysique et Psychologie. Par TH. FLOURNOY, M.D. 8vo. pp. vii., 133. H. Georg. Genève. 1890.

This little work, which appears to be an amplified edition, with notes, of the second of two "conferences," or lectures, on "Soul and Body," delivered in certain public courses under the auspices of the University of Geneva, in December, 1888, is an explanation and defence of the place which physiological psychology has won for itself, during the last thirty or forty years, among the positive and experimental sciences. The sitting at which it was pronounced was devoted, we are told (p. 52), "to determine the line of demarcation between positive psychology and metaphysic," a task of great interest, not for amateurs only, as the author modestly suggests, but also for those whose main business lies either in scientific or in philosophical pursuits, and moreover a task for which the lecture itself shows, that its author is eminently qualified.

Nothing can be clearer than the exposition which M. Flournoy gives of the way in which physiological psychology has become detached, as it were, from the philosophical group of sciences (so to call them), has asserted its autonomy, and has taken rank as the youngest of the positive and experimental sciences, a science possessing its laboratories and its instruments of precision, and proceeding to reduce its phenomena to measurement, which is the essential mark of all exact science. Nothing can be more definite than the author's statement of the two great principles, upon which, as he holds, this position is based, and by which it is justified. These principles are, first, the principle of concomitance or parallelism: "Every psychical phenomenon has a determinate physical concomitant" (p. 5); and secondly, the principle of dualism, or axiom of heterogeneity: "Body and mind, consciousness and molecular cerebral movement, the psychical fact and the physical fact, although they are simultaneous, are heterogeneous, disparate, irreducible [to one another], obstinately TWO" (p. 17).

I will endeavour to reproduce in English the paragraphs which lead up to this definition as their conclusion, seeing that they set the author's fundamental view in the clearest light: "How can two things so opposite as mind and matter, the physical and the moral, find themselves in relation and influence each other? This is what no one has yet explained, and the most subtile thinkers have spent in vain all the resources of their genius in trying to solve this irritating problem. Let philosophy at the

present day go on fathoming it—that is its business. But if psychology yielded to this temptation, all progress would for it be at an end. Psychology cannot advance, cannot even subsist as a science, save on condition of going straight on, without troubling itself with this mystery, any more than if it did not exist. Yet we are aware that it is no easy matter thus all at once to ignore a sigh of interrogation, which thrusts itself forward at every step. It is a ply to take; time is requisite. Accordingly it is well that, until the habit is acquired, the newly-born science should find in its fundamental principle itself a perpetual recall to order, and the warning that it has resolutely to turn its back on metaphysic.

"This warning is already contained in the very terms, *concomitance*, *accompaniment*, *parallelism*, and other equivalents, often employed to designate the principle in question. Just as two parallel lines, or two persons who are walking together, continue distinct and separate, and go on moving in company, without fusion and without collision, so, in the eye of experimental psychology, the series of psychical facts and the series of physiological events, while flowing on side by side, remain always different, distinct, without points of contact, without any conceivable mutual influence. They are simultaneous, that is to say, have a time-relation to each other, but that is all — no discoverable relation of action or of nature—they are heterogeneous.

"It is advisable, in order the better to bring out into prominence the important corollary of the principle of parallelism, to formulate it separately, as an independent truth, under the name of Axiom of Heterogeneity or Principle of Dualism." Then follows the definition which has been quoted above (pp. 16-17).

The first chapter having thus been devoted to set forth the essential principles of positive psychology, our author proceeds in chapter ii. to criticise the main classes of what he calls the metaphysical hypotheses, which have been propounded concerning the nature of the union between soul and body. These he reduces to four, namely, spiritualism, materialism, phenomenism or idealism, and monism. With these, however, and with his highly instructive criticism upon them, it is not here the place to concern ourselves. I propose similarly to pass over the discussion, with which his third and last chapter is occupied, on the position which science generally, and physiological psychology more particularly, hold with regard to the problems, first, of a future life, secondly, of free will and moral responsibility. Of the

first he says: "Its account is soon cast up—nothing for it, nothing against it" (p. 53). As to the second, his conclusion is, that, while no positive science can even entertain the question of liberty, determinism being its necessary pre-supposition as science, yet, seeing that determinism is not necessarily "the expression of absolute truth," it is beyond the true province of science either to affirm or to deny it, and that the attempt to do either the one or the other would at once rob science of its independence, and change it *pro tanto* into dogmatic philosophy. It is in short (in his view) a metaphysical and not a scientific question (pp. 83-84).

The rest of the book is occupied by notes, the greater part of which relates to the great principles expounded in chapter i. It is here that the main interest of the work is centred, at least for neurologists. It is the view taken of the constitutive principles of physiological psychology which determines what we are to think of the other matters which the book contains, or in other words, what relation we are to conceive psychology holding to the four so-called metaphysical hypotheses criticised in chapter ii. and to the questions of a future life and free-will discussed in chapter iii. It is, moreover, those constitutive principles of the new science, physiological psychology, which have most interest for men of science, and especially for neurologists, inasmuch as they determine its relation to its own better half, if so it may be called, namely, physiology. The following remarks will accordingly be addressed to the consideration of these two cardinal or constitutive principles.

And here the first and most prominent thing which strikes us is the author's open and repeatedly expressed dislike and contempt for metaphysic, founded, as will presently appear, on a misconception of what metaphysic really is. His attitude towards it is one of undisguised hostility. He speaks of "shaking off the yoke of metaphysic, by banishing from science the age-long quarrels of philosophers" (p. 6); he quotes against it a well-worn joke as to its utter unintelligibility (p. 7); every metaphysical system, he says, is well-known to have a false pass-key to the gate which opens on the Absolute (p. 20); he would give them all their final *congé* (p. 51); he speaks of the serene indifference of science in matters of philosophical belief (p. 53); metaphysic, he says, gives everyone exactly the answer he wishes, in a certain important question (p. 99); and, at p. 116, "We gladly make allowance for jugglery (*escamotage*) in persons who get their living by it, for instance in *la Métaphysique*, whom the force of circumstances has

always reduced to this extremity." Who can think that any one is likely to draw the line of demarcation truly between pyschology and metaphysic, whose views of the latter are of this nature? If mataphysic is jugglery, what need of 133 pages to demarcate it from science?

It is not to be supposed, that this feeling of hostility is anything like universal in the scientific world. It is probable that many men of science would gladly see an alliance between positive science and metaphysic, provided of course that a metaphysical system should be found possible, which is at once solidly based on facts of experience, has a method and principles of its own, and neither pretends nor attempts to impose *a priori* dogmas on any positive science. A metaphysical theory of this kind would probably be welcomed by many a man of science, as serving to complete the system of the positive sciences by showing what are the nature and limits of all speculative thought, upon whatever part of the field of knowledge it may be directed, including those of Ethic, Logic, Æsthetic, and Theology. Some further remarks on this subject will be made, in conclusion of the present article.

Now the establishment and recognition of physiological psychology as a positive and experimental science is, to say the least, as great a benefit to metaphysic, in the true sense of that word, as it is to psychology. It is at least as much the interest of metaphysic as it is of psychology, that the line of demarcation between them should be truly drawn. Metaphysic as much as psychology will be a gainer, by being definitely relieved of questions which do not properly belong to it, and which, while undistinguished and uneliminated, have been a perpetual source of confusion and embarrassment. We may therefore heartily concur with M. Flournoy in his wish to establish the true line of demarcation, though not concurring in his hostility to metaphysic. We may think with him, that it is the interest of science to be relieved of metaphysical questions; but we may think so for the additional reason that the metaphysician in every man, even in men of science, will thereby be most surely appeased and satisfied.

The whole question as to what the true line of demarcation is turns upon the sufficiency of the two great principles which M. Flournoy has laid down, concomitance and heterogeneity, first to constitute a positive science, secondly to keep it clear of all metaphysical matter. The constitution of psychology as a positive science on these two principles is M. Flournoy's main theme. The sufficiency of those principles for the establishment of a science free from metaphysic is therefore the cardinal point to which our

attention is directed. To the present writer it seems that their insufficiency is manifest, and that for both the purposes which they have in view. They neither suffice to constitute a positive science, nor do they exclude metaphysical conceptions, in M. Flournoy's sense of the term, from the science which they would constitute.

Let us begin with the principle of concomitance. By this is meant, as M. Flournoy repeatedly explains, that, between the two heterogeneous components, which together make up the total subject-matter of psychology—the neuro-cerebral process and the process of consciousness—no relation save that of simultaneity can be admitted by psychology to exist. Wherever there is consciousness, there some phenomena belonging to these two processes are found accompanying each other, in a regular and constantly recurring manner, and that is all that we know or can know about them; beyond this, whatever we may imagine of their relations is metaphysic.

Now to this narrow idea of what is requisite to constitute a positive science there is a most serious objection. Granting that the two series, the events of consciousness and those of certain neuro-cerebral processes, are totally heterogeneous, and their respective parts strictly and regularly simultaneous, this fact of simultaneity, concomitance, or parallelism, is not alone enough to weld them into the subject matter of a single science. Simultaneity roughly observed may have been the preliminary step, the first incentive, towards a science; but the science is not constituted until the relation of dependence is added to and combined with that of mere concomitance. It is the dependence of one phenomenon on another, whether reciprocal or otherwise, that all positive science seeks to establish. The idea of dependence, or of real conditioning of one thing by another, is the root idea in which science itself, as a mode of knowledge, takes its origin. Let us hear M. Flournoy himself on this point: "What is it, in fact, to *know* an event, to understand it, to make it an object of science, but to connect it with its causes, that is to say, to assign the series and the complete grouping (*l'ensemble*) of antecedent events which have produced it, which have rendered it necessary? To explain a fact is always to refer it to other facts in which it was implicitly contained, and in virtue of which it could not but be, it could not be otherwise than it is" (p. 64.)

In point of fact, the very same observations which show the concomitance of the two series of events, physiological and psychical, show also the dependence of the psychical on the

physiological, in respect of their coming into and continuing in existence. In natural or rather in non-artifically instituted processes, as for instance in external sensation, we find the sensation dependent upon, as well as concomitant with, neural processes which are set up directly by the physical environment; we never find sensations setting up the neural processes which are concomitant with them. Take away the neural process, and there is no sensation. Take away the sensation—it cannot be done, save by taking away the neural process. There is therefore dependence of the sensation on the concomitant neural process, but not *vice versâ*.

The same reasoning must be taken to hold good also of intra-cerebral processes and their concomitant psychical events or states of consciousness, I mean such as association, thought, volition, and emotion; though since here we have not the external agencies, which directly set up neural motion, to appeal to, the demonstration of the fact is far more difficult, and the anticipation of it is still awaiting its complete experimental justification. Its truth, however, is not precisely what I am now concerned to prove. I assume, with M. Flournoy, the thorough-going concomitance of the two sets of phenomena, and then I say, that the same reasoning, which justifies the assumption of concomitance, also justifies and renders necessary the assumption of dependence.

Again, in artificially instituted processes or experiments, we have no other means of laying hold of, producing, or so to speak bringing into court, states of consciousness, save by setting up physical changes which act upon the neural or neuro-cerebral system. Here again, whenever we have reason to infer concomitance, there we have reason to infer dependence also. Indeed it is only by means of the dependence that the idea of a precise concomitance is arrived at; seeing that exact simultaneity, that is, the precise coincidence of beginning and end, between a psychical event and the neural process which it accompanies, is matter of supposition and definition, and not a fact which is capable of being directly observed. By dependence I mean of course dependence of psychical phenomena on physiological, but not *vice versâ*. There is thus no fear of our being led to suppose "a mixed chain in which purely psychical events (sensations, desires, volitions, &c.) should come to be intercalated between physical events (centripetal or centrifugal innervations)" (p. 86). Of the two heterogeneous and concomitant series, the psychical is dependent on, as well as concomitant with, the physiological.

And what is more, this fact seems to be tacitly assumed and counted on throughout M. Flournoy's book, though in express words he repudiates it.

The main reason which he alleges for repudiating it is found in his second constitutive principle, the principle or axiom of heterogeneity. "Since it is just the existence of a bond (*lien*) between two things so heterogeneous which is incomprehensible for our intelligence, it is better to make no allusion to it" (p. 86). But the existence of a bond between physical events taken alone (or for that matter, between psychical events taken alone), is just as incomprehensible for our intelligence, as one between physical events and psychical. Cohesion, attraction, chemical affinity, for instance, are in their ultimate ground or reason equally beyond our power to conceive; the so-called bond between the facts or events which we refer to them is equally incomprehensible; and why? Because, taken alone, it is a name hypostasised and nothing more; no bond other than what is included in the facts or events themselves exists. The terms just cited are names for facts, for events, for phenomena connected in time and space, which we either perceive, or else infer from perceptions, in cases which are beyond the range of our direct perceptive power; and similarly the connection between physical and psychical events is an observed fact, the ultimate ground or reason of which we cannot penetrate. But we do not hesitate to describe physical events as dependent one on another. In their case the *fact* of their dependence suffices us. So also it should suffice in the case of psychology. The heterogeneity of the phenomena between which it is observed adds nothing to the supposed incomprehensibility of its ultimate ground or reason; for that incomprehensibility is in all cases alike at a maximum.

If it should be replied, that after all what we really know of dependence is reducible to the co-existence or succession of the phenomena concerned in time and space, and that this is what is meant by calling it *concomitance* and nothing more, the answer is, that this is equally and alike true of physical phenomena taken alone; that notwithstanding this, we can and do distinguish between cases of dependence and cases of mere simultaneity, or concomitance, without dependence; and that the establishment of the fact and laws of dependence is the sole purpose of science. To science the fact of mere simultaneity is always an *explicandum*, and not an *explicatio*.

A simple and well-worn instance will make this clear. As the earth revolves on its axis in presence of the sun, it turns opposite

segments of its surface successively to the sun's light and heat. As one segment of its surface comes into the light of day, the opposite segment comes into the darkness of night, and so on perpetually. The two phenomena, of one segment coming into one position and the opposite segment coming into the other position, are strictly concomitant or simultaneous, but there is no dependence of either on the other. On the contrary, both alike depend upon the physical fact (which is also concomitant with the whole succession) of the revolution of the earth on its axis in presence of the sun. That is the fact which it has been imperative on the science of astronomy to ascertain.

But this does not exhaust the instructiveness of this simple instance. It deserves dwelling on a little longer. The light of day and the darkness of night are sensations. They belong to M. Flournoy's psychical series of phenomena, as distinguished from his physiological series. As sensations there exists between them the relation of contrast. As sensations of a simple individual, there is between them the relation of successive alternation in time-sequence. As sensations thought of, by the psychologist, as being experienced by differently placed individuals, they stand in the relation of simultaneity. Now what has all this to do with the interests of psychology? Simply this, that it is something for psychology to explain. And how? Why by ascertaining, first, the dependence of the sensations in question on states of the neuro-cerebral organism, and secondly, the dependence of these states on the ethereal vibrations with which it comes into contact. M. Flournoy seems to suppose, that there can be no dependence of one phenomenon on another, unless the phenomenon called dependent reacts upon that on which it depends. But for this notion, in the case of psychology, there does not seem to be the smallest warrant. All the ascertained facts indicate dependence (as well as concomitance) of psychical on physiological phenomena, without a trace of any reaction of the psychical on the physiological.

What, then, it may be asked, do you precisely mean by the dependence of some phenomena on others? The answer is—dependence means a relation such that, given the conditioning phenomenon, the dependent phenomenon invariably occurs, and in the absence of the conditioning phenomenon, the phenomenon called dependent invariably makes default. The idea that, to establish the fact of dependence between phenomena, we must have a knowledge of what constitutes a bond or *lien* between them, is an idea which has long been given up in the physical

sciences. To retain it, as M. Flournoy does, in the new science of physiological psychology is retrograde, and not retrograde only —it is suicidal.

For observe, in basing psychology on the mere concomitance of its two sets of heterogeneous phenomena, excluding the fact of the dependence of the psychical on the physiological, M. Flournoy not only narrows its foundation, in contravention of positively observed facts, but he robs it of its rank as a positive science. Previous to the rise of physiological psychology, there were two claimants in the field for the office of being the really conditioning agent or agency in generating, sustaining and governing the phenomena of consciousness. One was the soul, mind, or spirit; the other was the ego or some other form or mode of consciousness itself. For, as remarked above, all science aims at ascertaining the laws of the real dependence or inter-dependence of phenomena. The value of physiological psychology consists, therefore, in the fact that the neuro-cerebral system is a new and rival claimant for this office. If it comes into the field with the disclaimer of any intention to entertain the question of the real conditioning of the phenomena of consciousness, it not only abdicates its claim to be a positive science, but it leaves the field open to the former rival claimants, and that to the great detriment of metaphysic, which is thus left to contend single-handed against those formidable adversaries. Physiological psychologists really ought to have the courage of their opinions.

This leads us to the second of the two failures, the two insufficiencies, noted at the outset, of M. Flournoy's great principles of psychology; I mean their insufficiency to keep psychology clear of conceptions and questions which M. Flournoy himself would designate as metaphysical. At page 118, he quotes a long passage from Professor Wundt's "Essays" (Leipzig, 1885), p. 115-116, which ends with the following sentences (I translate from M. Flournoy's French translation): "Accordingly the naturalist, who, in his own domain, treats the world with justice as a reality independent of our mental phenomena, can never lay down anything beyond a *parallelism* between these latter and the concomitant physiological functions. This parallelism involves the supposition that the phenomena of the psychical life form of themselves a closed totality (*un tout fermé*), a chain of causes and effects as complete as that which exists in the material world; but just because we are thus in presence of two causal series, each complete on its own account, there can be no passage from the one to the other."

To this view M. Flournoy hastens to give his unqualified adhesion in the very next paragraph, which concludes as follows: "To psychical facts we must assign psychical causes and effects; to physical facts physical causes and effects; and never seek in one of these spheres the *cause* or the *effect* of that which takes place in the other (unless we take these words not in the strict but only in the figurative sense, as meaning simply the *concomitant*)" (p. 118-119).

Here then, it is to be supposed, we have M. Flournoy's real meaning; at last the secret is out. Now we see what is meant by his so modestly restricting physiological psychology to a concomitance (excluding dependence) of two sets of heterogeneous phenomena. The fact of dependence (commonly called causality) which is excluded from the relation between neuro-cerebral process and consciousness, is admitted between the phenomena of each sphere taken by themselves. But these spheres or sets of heterogenous phenomena together constitutes the subject-matter of physiological psychology. The fact of dependence, therefore, though so carefully excluded from the relation between the physiological and the psychical, nevertheless comes into the subject-matter of physiological psychology, and that in two places instead of one. And since the psychical states, between which it is admitted, are an essential part of the subject-matter of the science, the whole question concerning the genesis of, and nexus between, psychical states is not only re-opened, but is also re-introduced into physiological psychology, and that with the additional announcement that physiology can contribute absolutely nothing to its solution. In short, physiological psychology, in M. Flournoy's hands, simply and expressly abdicates in favour of what M. Flournoy would call metaphysical hypotheses concerning the nature of the soul. As to his proposed line of demarcation between physiological psychology and metaphysic, it vanishes completely and irrecoverably.

Moreover, the idea of a causal nexus between psychical phenomena taken alone—an idea which, as we have seen, Professor Wundt expresses by calling them "a closed totality, a chain of causes and effects,"—completely breaks down when applied to the facts. To revert to this simple instance of the light of day and the darkness of night, which as already stated are strictly psychical phenomena, it is impossible to suppose that the light of day is caused by the darkness of the preceding night, or the darkness of night by the light of the preceding day. And equally impossible is the supposition that either of them is caused by the psychical

events which have taken place in the minds of those who have lived through the day or the night preceding them—that the dreams, say, of sleepers are among the causes of morning. Obviously their cause is partly of a physical, partly of a physiological nature. The "closed totality" theory of psychical events is wholly inapplicable to explain the fact of their occurrence.

This deplorable muddle, for so it must be called, is due solely to M. Flournoy's determined exclusion of the fact of dependence from the idea which he forms of the concomitance or parallelism between neuro-cerebral processes and consciousness. He intends thereby, no doubt, to set physiological psychology free from even the semblance of admitting metaphysical matter. But inasmuch as it is a departure from the simple truth of observed facts, it is sure, sooner or later, to issue in more serious and more manifest error. The most singular thing about it is that the idea of dependence is not strictly, as M. Flournoy seems to imagine, a metaphysical, but a scientific idea. In admitting it into his conception of the constitutive principles of physiological psychology he would not have ventured upon metaphysical ground at all, but would have been keeping strictly within the domain of positive science.

M. Flournoy seems to share the view too commonly taken of metaphysic, namely, that it consists of guesses at the solution of problems which have been abandoned by science as insoluble, and in the form which science, before abandoning has given them. If this were so, it is needless to say that the contempt which M. Flournoy expresses for metaphysic would be fully justified. But in reality nothing can be farther from the truth than this idea. Metaphysic is the analytical examination of the knowledge we have of things, both physical and psychical, in its character of knowledge instead of being (as science is) an examination of things physical and psychical in their character of given objects. It thus deals with the very same phenomena as positive science, but it deals with them on their subjective side, which is but varying the phrase used above, in saying that it deals with our knowledge of things physical and psychal. Now dependence, or what is commonly called causality, belongs only to things as given objects; it is the real order *of their occurrence*, both in co-existence and in sequence, expressed as subject to constant laws; it is therefore a scientific and not a metaphysical idea; to ascertain this order in all departments of the course of nature is the whole business of science. What metaphysic has to do with it is merely to ascertain what our knowledge of it is, what the

meaning of the term is, and what evidence we have for its reality in that meaning. The only sense in which it can be said that metaphysic goes beyond, or is the completion of science, is this, that it gives the obverse and subjective side of the same phenomena which science treats on their objective side, as things or objects already given and existent. It in no way attempts to give a more complete scientific answer to scientific problems than science itself can give.

Psychical states and events, which as we have seen, are one half of the subject matter of physiological psychology, are no exception to the universally applicable distinction just laid down. They also, like physical things, can both be treated as given objects, and have the laws of their occurrence ascertained, as they are ascertained by psychology, and also be treated as pieces of knowledge or experience, that is, as they are actually known or experienced in consciousness, consciousness being itself a process of knowing. If, therefore, it is only as given objects, dependent on other given objects, that they come within the scope of science, it follows that the laws of their genesis, evolution and combination, as so depending, are what a scientific psychology has to investigate and ascertain.

But if this be a true account of the relation between metaphysic and positive science, it is obvious that, so long as this relation is kept in view, there can be no clashing between the two pursuits, not even when the phenomena treated of are those of consciousness. Metaphysic and psychology have each its own method, its own purpose, and its own principles, in dealing with them. Metaphysic treats them as part and parcel of our total knowledge of the Universe about us, psychology treats them in respect of their connection with the organisms, to which they belong. Nor does this distinction relate only to those states of consciousness which are our knowledge and evidence of an external and material world; but the states of consciousness which constitute what are called the phenomena and laws of Ethic, Logic, Æsthetic, and Theology, also fall into the province of metaphysic so far as their nature and validity are concerned, and into the province of psychology with respect to their genesis in the neuro-cerebral organism.

The line of demarcation between physiological psychology and metaphysic is drawn clearly and precisely by means of the above distinction. And in consequence of it the two pursuits are placed on the footing of friendly allies, all reason for dispute and hostility being removed. The basis of the alliance is found

in the simple fact, by which also the above distinction is justified, namely, that the whole of the *causæ cognoscendi* of all phenomena whatever lies in consciousness—the whole of their *causæ existendi*, so far as we have any means of positively tracing it, lies in matter. If there were no consciousness, matter could not be known to exist; if there were no matter, consciousness could not exist in human organisms. Physiological psychology rightly takes both consciousness and organisms as given existents or realities; but it is false to its own principles if it denies, either that the relation between them is one of dependence, or that this dependence is the dependence of consciousness on the organism. If facts should be observed which compelled us, in theory, either to reverse this order of dependence, or to admit that, though there was simultaneity there was no dependence, then there would be an end of physiological psychology.

SHADWORTH H. HODGSON.

Des Phénomènes de Synopsie (Audition Colorée); Photismes; Schèmes visuels; Personnifications. Par TH. FLOURNOY, M.D. Paris: F. Alcan, 1893.

In this volume Prof. Flournoy gives us the results of inquiries carried out partly by himself, partly by a student of medicine, M. E. Claparède, into the curious phenomena known as coloured hearing and allied phenomena. These have already been dealt with by Bleuler and Lehmann, P. Galton, Grüber and others. At first the range of phenomena investigated was limited to vivid images of particular colours immediately excited or concomitants of particular sensations of sounds, articulate and musical. This field has gradually been enlarged by the recognition of a similar excitation of visual images or photisms by other than auditory sensations, and also of the excitation of uncoloured visual images, as those of particular schemes and diagrams in connection with the series of digits, days of the week, months, &c. Prof. Flournoy still further extends the area of phenomena by including what he calls personifications, that is, the suggestion of particular types of human face or complete figures, by the numerals I., II., III., &c. To these phenomena collectively, he gives the name of visual synthesis or "synopsie." The setting forth of these various divisions of synoptic phenomena is contained in chapter i.

The next chapter discusses the psychological principles by which it is proposed to account for the phenomena. These are

reduced to three: of these the first is affective or emotional association; that is, similarity of emotional tone or analogy of feeling, as when a high musical note excites the visual image of a bright colour. The second is habitual association, or the effect of repeated presentation together (association by contiguity), as when the months and the days are represented in columns resembling those of an almanack or calendar. The third principle is privileged association, by which is meant the effect of specially interesting and impressive juxtaposition, especially in early life. In the nature of the case these associations, dating from a remote period of life, cannot in general be certainly ascertained. The author recognises that it is as yet practically impossible to say how much is referable to the action of one principle, how much to that of another. He tells us that he has been unable to find a subject who can indicate with exactness the origin and cause of his photisms.

Chapter iii. begins the detailed examination of the phenomena by an account of alphabetical photisms, *i.e.*, the visual images suggested by particular vowels and consonant sounds. A large proportion of those who have coloured hearing at all have it in connection with vowel sounds, whereas, only about one-fifth of those who hear the vowels coloured attribute colour to consonantal sounds. Coloured hearing seems to be a very variable capacity, some displaying it more widely with reference to letters, sounds, days, &c., others more narrowly with reference only to particular excitants, as certain letters, certain sounds, &c., and some exclusively with reference to one letter, one month, &c. The complexity of the excitant (inducteur), *i.e.*, the auditory, visual, articulatory and manual (scriptive) elements in letters and words is then discussed. Thus the various degrees of intensity of the indexed visual image are distinguished, as they range from localised hallucinatory quasi-perceptions through images of varying degrees of vividness down to vague non-pictorial thoughts. With regard to the photisms excited by vowel sounds it is shown that amid wide diversities of individual experience there is observable a certain amount of constancy or uniformity. This applies to the particular hist attributed to the vowel, as also to the degrees of brightness of the colour. Thus, while 52 persons see the *a* as white, only 4 see it as gray, while 50 see it as red, only 3 see it as green. With respect to brightness the amount of agreement is much larger. Thus, while 114 persons visualise the vowel *i* as bright, only 18 visualise it as dark. Photisms excited by the diphthongs, the consonants, and by whole words are dealt with in a similar detailed manner.

In chapter iv. the writer gives an account of other photisms, viz., those induced by musical sounds, by odours and tastes, &c. The notes of the diatonic scale appear each to have its proper colours to a considerable number of the subjects examined, but there is great diversity as to what particular colour characterises a given note. Thus the third (mi) appears now as rose, now as blue, and so forth. The colour-photism seems to be affected by the timbre of the tone. As to brightness there is a general agreement that high tones are bright, low tones dark.

These musical photisms appear to be largely due to affective association; yet in certain cases the action of (contiguous) association is traceable as when a tone is given the same colour as that of the instrument producing it, *e.g.*, when the tone of a piano is seen as white and black, that of a violin as the colour of the wood. Notwithstanding the well-known power of odours in exciting vivid recollections, these sensations as well as tastes but very rarely induce photisms. Under the head, "Photisms of sympathy," we have described the curious fact that in the case of at least one subject persons who are antipathetic excite a disagreeable colour (violet), whereas those who are sympathetic excite an agreeable colour (rose or red). Names of persons, places, days of the week, &c., appear also to excite photisms in a certain number of cases. The same is true of the numerals, 1, 2, &c. The chapter concludes with a reference to non-visual synæsthesis, as when the tickling of a certain part of the back of the left hand provokes a disagreeable sensation on the left cheek which produces a grimace and a movement of the hand to the spot. But one notices here the want of a recognition of the large area of such "Mitempfindungen," as described by Lotze and others.

The two chapters on schemes and diagrams add interesting details to the account given by Galton. Very curious is the series of increasing and decreasing ovals or circles corresponding to the succession of the months. In certain cases, as that of the French novelist, Pierre Loti, a month assumes a more concrete or pictorial symbol. The diagrammatic representations of numbers, days of the week and months are very various, but are too complicated to be described here. These geometric arrangements are in many cases definitely externalised and localised, as before the subject, above his head, in a definite portion of the visual field. The writer shows that these diagrams are useful as giving intuitive clearness to the mental representation of time sequences. The origin of the diagrams is thus explained tentatively by means of the principles given above.

Chapter vii. describes personification, that is, the tendency in certain persons to picture numbers, names of the months, &c., under a definite concrete form, as when 1, 2, 3, are said to be children not definitely characterised playing together.

The volume concludes with a chapter on the evolution of "synopsies," in which the writer traces out the variations in the phenomena with age, and discusses the question whether they ought to be classed as pathological. He decides that while abnormal, they cannot be called pathological. A supplementary chapter is added, dealing with the case of M. Grüber, who sent a memoir on the subject to the Congress of Experimental Psychology, held in London in August, 1892, and is engaged in making further statistical inquiries.

This slight sketch of Professor Flournoy's volume may suffice to show that it is an interesting contribution to the study of some of the most curious psychical phenomena lying on the confines of normal and abnormal life. As such it deserves the attention of psychologists and pathologists. What is most needed in the present stage of the inquiry is perhaps a wide statistical examination of children. It seems highly probable that, as in the case of a girl known to the present writer, coloured hearing and similar phenomena are often clearly marked in the early years, and disappear with adolescence. It would also be important to find out to what extent the phenomenon accompanies susceptibility to visual hallucination, to vivid imagination generally, as noted, say, by Galton's questions.

JAMES SULLY.

Critical Digest.

HYSTERIA AND NEURASTHENIA.

BY J. MICHELL CLARKE, M.A., M.D., M.R.C.P.
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I.—Pathogenesis of Hysteria and Neurasthenia.
II.—Symptoms, &c., of Hysteria.
III.—Hysterical and some other Functional Disorders of Movement.
IV.—Hysterical Disorders of the Organs of Vision.
V.—Hypnotism, Hysterical Somnambulism, and Double Consciousness.
VI.—The Association of Hysteria with Organic Disease of the Nervous System.
VII.—The "Traumatic Neuroses."
VIII.—General Features of Neurasthenia, after Löwenfeld.
IX.—Treatment of Hysteria and of Neurasthenia.

"Pathologie und Therapie der Neurasthenie und Hysterie," dargestellt von Dr. L. Löwenfeld, Specialarzt fur Nervenkranheiten in München. 2 vols. J. Bergmann, Wiesbaden, 1894, pp. 744.

"Hypnotisme et double Conscience, origine de leur étude et divers travaux sur les sujets analogues," par le Dr. Azam, Prof. hon. à la faculté de Médecine de Bordeaux, &c., avec des préfaces et des lettres de MM. Paul Bert, Charcot et Ribot. Paris, Felix Alcan, 1893, pp. 375.

"La Névrose Traumatique. Etude Médico-légale sur les blessures produites par les accidents de chemin de fer et les traumatismes analogues," par le Dr. Ch. Vibert, expert près le tribunal de la Seine. Paris, Libraire J. B. Baillière et Fils, 1893.

"État Mental des Hystériques. Les Stigmates Mentaux," par Pierre Janet. Paris, Rueff et Cie., 1893, pp. 233.

References to other pamphlets and papers abstracted will be found at the end of this digest.

I. Papers on the Theory of Hysteria and of Neurasthenia.

I.—Dr. Löwenfeld's work is the most important recent contribution to the literature of hysteria and neurasthenia. It is clearly written, in an interesting style, and the author is thoroughly conversant with the most recent advances in the knowledge of these neuroses. It is, in our opinion, the best work on the subject. It is marked by originality of thought, soundness and independence of judgment. The author has done full justice to the works not only of German writers but of those of other nations, his criticisms are conceived in a broad and liberal spirit, and his own experience enables him to form a just estimate of the observations of other workers in this field. In the following pages a somewhat full account is given of the section on neurasthenia because there is much information in this section which is either not to be obtained elsewhere, or exists only in scattered papers. On the other hand, with regard to the section on hysteria, only those parts of this section which contain novel matter, or deal with points which are still a subject of dispute are here considered. It is not to be inferred from these necessarily fragmentary references that the account given of hysteria in this book is otherwise than full and complete, and indeed, in all respects a most valuable one.

In commencing his book with some general considerations, Dr. Löwenfeld observes that hitherto in spite of numerous efforts, we know of no anatomical changes as the cause of either neurasthenia or hysteria. Clinically it is difficult to separate the essential and distinctive from the non-essential symptoms of neurasthenia, and to isolate a distinct type for it. In hysteria, the numberless attempts to get a satisfactory definition, and to bring its symptoms into a definite form, have failed.

There are two chief views on the place of hysteria amongst nervous diseases—that it is (1)a general neurosis, a disorder of the whole nervous system, or (2) a psychosis, a disorder of the organ of perception, *i.e.*, of the grey matter of the brain. This latter view has been pushed furthest by Möbius, who observes that hysteria comprises all those morbid changes in the body which are brought about through representations (ideas). If this conception is adopted, then hysteria depends on a functional disturbance of the cortex, and further, a psychical therapeusis is the only rational mode of treatment for it.

Such a dependence on idea, or on psychical processes in general for all hysterical symptoms is not proved, nor even shown

to be probable; an analogous mode of reasoning would be that, because influenza is often the cause of neuralgia, therefore all neuralgias arise from influenza.

The origin of hysterical symptoms from idea, is, in the author's opinion, only true for a limited number of them, and the hypothesis of the "idiogenesis" of symptoms is not an "open-sesame" to the secrets of all hysterical processes. If the idea of inability to move a limb can cause paralysis, the cerebral process in question consists of an inhibition of the centres concerned in the movement by this idea of powerlessness, so that other stimuli can no longer affect them. But if severe hysterical convulsions with a possibly fatal issue, attacks of profound syncope or fever can be produced through idea, then the cerebral processes connected with such symptoms defy interpretation; in these cases knowledge of the cause fails to explain the results. There must be a pecular condition of the nervous system to render such consequences possible, and it appears to the author quite immaterial whether this condition is produced by idea or any other cause whatsoever. The effects of a gunpowder explosion depend on the quantity and quality of the powder, and not on the composition of the spark which ignites it. The altered condition of the nervous system is a *sine quâ non*, and is of so great importance that the exciting cause of the symptoms sinks into insignificance.

The fact that in hysteria psychical agencies may cause functional disturbances, which in health could not be similarly aroused, is not enough to constitute hysteria a psychosis. The special disorders which occur in hysteria are not related so much to anomalies in the formation of ideas themselves, as to their effects, connected as these are not only with the condition of the grey cortex, but also with abnormal irritability of the subcortical centres. Further psychical disturbances in hysteria, with the exception of delirium are not of such a kind, nor so constant, as to warrant it being considered a psychosis. There is no proof that the development of the hysterical disposition depends entirely, so far as the central nervous system is concerned, on abnormal conditions of the grey matter. The predisposing causes of hysteria are, in great part, of such a nature as to affect the whole nervous system. If hysteria is not a psychosis and is not related only to changes in the cortex, but also to changes in the lower centres, it follows that the author allies himself to the older view that hysteria is a general neurosis, and that its fundamental character is, as is the case in neurasthenia, a state of irritable weakness in which, as a rule, irritability is in excess of weakness.

Nervousness.—Transitional forms connect hysteria and neurasthenia with other neuroses and many pyschoses as well as with conditions which cannot be considered as distinct disease, namely, those which are entitled from the point of view of symptoms as "nervousness" or irritable weakness, and from that of etiology as the neuropathic constitution or disposition. "Nervous" and "nervousness" are used by the public as convenient terms to express a variety of conditions, and, unlike that of hysteria, convey no sense of reproach. The most severe and the lightest affections in the domain of neurology are alike called "nervous." Science cannot make use of these terms in the wide sense that custom has given to them, and it is necessary to inquire what is to be definitely understood and defined by "nervous" and "nervousness." Under certain conditions—in the wear and tear of ordinary life—a sound man may become nervous; in some circumstances not to be nervous would prove the possession of an abnormal or unusual condition of the nervous system. To this transitory and, so to say, physiological nervousness stand opposed the permanent, life-long, frequently congenital states of nervousness. In the latter, increased reaction to external agents in the form of psychical disturbances is of chief importance. Irritations which produce trifling effects in the healthy, give rise to disagreeable and even painful sensations, accompanied by motor, vasomotor and secretory reactions. Emotional processes produce more easily far greater effects on the whole central nervous system than in the healthy.

With increase of sensibility occur the symptoms which point to weakness and excessive irritability of the functionating centres. The facts at command do not allow this increased irritability and lessened power of work to be wholly localised in the same elements or parts of the central nervous system. Lechner explains such symptoms by a disturbance of the normal relation between muscle and nerve-irritability. That the exhaustion of nervous persons can in no way be wholly or even generally imprinted on the central activity is shown by the fact that there is often no diminution of the intellectual powers. The author enumerates the different symptoms by which nervousness is shown in the child and in the adult, and points out that strong, well-built men are affected by it as well as those of more delicate organisation; that under favourable circumstances good, often first-rate work is done in the fields of art, science and industry by such individuals, and that the condition is to be regarded as one midway between health and disease. In favourable surroundings the

nervous nearly attain to good health, but under unfavourable ones—prolonged strain and anxiety—a farther development of the condition in the direction of disease occurs, and the symptoms of hysteria or of neurasthenia, that is to say, of pronounced disease, appear. Neurasthenia and hysteria do not always, however, thus develop out of an antecedent nervous condition, they may be directly excited by severe shocks, either physical or mental, such as occur in railway accidents.

Distinction between Hysteria and Neurasthenia.—If we inquire how the distinction between neurasthenia and hysteria originated, seeing the similarity of their basis and fundamental characters, the reply is that it has arisen rather in the mode of growth of our knowledge than in the nature of these affections. The chief characters of hysteria have long been recognised; the symptoms of neurasthenia, until Beard in recent times accurately described them, were confounded with those of very different conditions. Curious distinctions between the two affections have been drawn in recent years, but the physician who is concerned not with the theories of disease, but with the actual sufferers from it, learns by experience that hysterical symptoms often occur in typical cases of neurasthenia and *vice versâ*, and that the distribution of the two neuroses in great part overlaps. There is certainly justification for the use of terms such as neurasthenic-hysteria or hystero-neurasthenia, and many authors attribute no great importance whether in one or the other case the designation neurasthenia or hysteria is used. The conception laid stress on by French writers that neurasthenics with hysterical symptoms and hysterics with neurasthenic symptoms are suffering from two different disorders at the same time seems far fetched. After all, our present knowledge indicates the resemblance of hysteria and neurasthenia in essential points, with varying alterations in the intensity and in the extent of the disturbance of the nervous system.

In numerous neurasthenical and in many hysterical persons, a melancholic or hypochondriacal disposition is met with; when this predominates over the other symptoms, we no longer speak of hysteria or of neurasthenia, but of hypochondriasis. Under the last title are included different conditions—hypochondriacal psychoneurosis or hypochondriasis in the strict sense of the term, hypochondriacal melancholia and monomania. The two last forms of psychical disturbance are distinguished by the intensity and persistency of the melancholic disposition and by the pre-

sence of hallucinations. As regards the hypochondriacal psychoneurosis, Gugl has lately insisted that all those cases are to be considered as hypochondriasis in which the mental distress is altogether out of proportion to the actual ailments. The author only recognises the influence of hypochondriasis in so far as the psychical factor—hypochondriacal imaginations and feelings—dominates over the other symptoms.

Theory of Neurasthenia.—Meynert explains the symptoms of neurasthenia as due to the reciprocal influence of irritability and weakness of particular parts of the brain in inducing change in one another. The nutrition and irritability of the cortex and of subcortical areas respectively, may be very different in the same parts of the brain at the same time, and in weakened heart-action the nutrition of the subcortical fails more readily than that of the cortical centres. Long before disturbances occur in the cortex itself, a weakening of the cortical centres is shown by loss of their inhibitory influence on the subcortical, projected into the hemispheres in the form of hallucinations and convulsions. Thus the activity of the subcortical centres, and of subcortical vasomotor centres is increased, gives rise to hyperæmia of the cortex, and hence to irritable weakness of localised parts of it. Meynert further considered the irritability of nerve-cells as a function intimately bound up with their nutrition, and consisting in a molecular attraction on the surrounding plasma. The psychical element in "nervous shock" consists in the loss of this molecular attraction, and these molecular changes are not to be considered as altogether mechanical, but as chemical (nutritive), being liable to take place rapidly on account of the instability of the phosphorus compounds.

Kowalewsky relies on some experiments of Anfimow and Sadowsky, who showed that long-continued electrical irritation of peripheral nerves induces structural changes in central nerve-cells (coagulation-necrosis and vacuolation). Excessive work of nerve elements causes (1) defective nutrition of their protoplasm; (2) auto-intoxication (*a*) from increased katabolic, and (*b*) from insufficient anabolic processes. Accompanying this auto-intoxication there is general insufficiency of tissue-oxygenation, and retrogressive blood-changes which further damage the nerve-elements, and through impoverishment of the blood interfere with general nutrition. Hereditary neurasthenia is attributed to poisoning of the central nervous system with ptomaines and leucomaines from hereditarily defective processes of metabolism.

Lechner holds that neurasthenia results from disturbance of the normal relationship in action between muscle and nerve. Dr. Löwenfeld observes that neurasthenia would be better understood if the changes which take place during the stage antecedent to physiological fatigue were better known. The changes in the nervous system in neurasthenia must be molecular, and are never gross. During the waking state full restoration of used up material and removal of the products of the activity of central nervous system does not occur. If the nervous capital is to remain the same, it must be restored during sleep. In neurasthenia the accumulation of waste products is so great that the period of sleep is no longer sufficient to fully remove them, so that a chronic over-lading of the nervous system takes place. In this way a neurasthenic condition is produced, and Kowalewsky's theory holds good for a great many cases, but the occurrence of neurasthenia from physical or mental shocks, from insolation and poisoning cannot be thus explained. If, however, a severe commotion of the nervous elements can reduce their metabolic activity, an accumulation of toxic products may be supposed to take place.

These disturbances of chemical tissue change in neurasthenia affect different parts of the central nervous system in different persons, but all parts are so bound together that one centre cannot be disturbed without affecting another. Vasomotor disturbances, started by slight and normally inadequate causes play an important part in the symptomatology of the disease. Lastly, the signs of increased irritability of nerve centres are not merely due to diminished inhibition from higher parts, but to an original abnormal increase of sensibility to external influences.

Dr. Joseph Breuer and Dr. Sigm. Freud[1] have been struck with the importance in cases of hysteria of ascertaining the causes and the phenomena immediately preceding the onset of the hysterical symptoms. Partly from the patient's forgetfulness or reluctance to recall a disagreeable experience the most thorough examination generally fails to elucidate these points, and it is necessary to arouse in the patient's mind by means of hypnosis the memory of the symptoms experienced at the onset. Then the connection between the latter and the symptoms present at the time of examination comes out in the clearest and most convincing manner. Observations made in this way are worthy of record, both from the theoretical and practical points of view.

In traumatic hysteria the connection between the exciting cause and the resulting symptoms is evident, and it is clear also in cases of hysterical attacks in which the same prodromal hallucinations, derived from the patient's surroundings, appear in each attack. The authors' experience is that an equally close connection with the exciting cause exists in the various symptoms which pass for spontaneous or idiopathic manifestations of hysteria, and that all varieties of hysterical phenomena (*e.g.*, neuralgias, epileptiform fits, paralyses, contractures, anæsthesias and special sense affections) can be referred to similar ætiological factors. The disproportion between the cause—acting for once only—and the resulting permanent symptoms is quite as marked as in traumatic hysteria; often the disturbing event has happened in childhood. Sometimes the connection between symptoms and cause is as clear and direct as possible, in other cases it is not so definite, but consists in a symbolic relation, so to speak, between the cause and the pathological phenomena, such as the occurrence of neuralgia or disgust for food as the consequences of moral pain and loss of appetite respectively. In other symptoms, again, *e.g.*, hemianæsthesia and contraction of fields, no such connection can be traced.

Such observations appear to show, as regards the pathogeny of the cases, the analogy of ordinary hysteria to the traumatic neurosis, and to justify an extension to the former of the current conception of traumatic hysteria. Psychical shocks (traumata) act as the causes of many, if not of most hysterical symptoms. Any experience which entails emotions of shame, terror, dread, &c., may be such a cause, its potency depending entirely on the sensibility of the person affected. Further, in ordinary hysteria, in the absence of more powerful agencies, a series of minor shocks may by summation be a potent cause. Again, conditions, which acting alone would be powerless, may produce hysterical symptoms if they happen to act in conjunction under favourable circumstances.

The causal connection of the physical shock with the resulting hysterical phenomena is not merely in the French sense one of "*agent provocateur*," after whose action the phenomena are capable of further development. The shock and the remembrance of the shock is rather to be considered as a "foreign body," which can still be potent a long time after its occurrence as an exciting agent. Proof of this is found in the fact that if the remembrance of the original cause or causes of the symptoms can be clearly aroused, the emotion which at the time

of onset accompanied them is also called up in the patient's mind and that if the patient then pictures to himself as fully as possible what took place, and describes his sufferings aloud, the hysterical symptoms described disappear and do not return. All varieties of hysterical symptoms may be treated in this way. The authors consider that the efficacy of this method of treatment lies in the patient's giving expression to his sufferings, and not in a suggestion arising from his expectation of being cured by the procedure. They conclude that the exciting causes of the events immediately prodromal to the onset of hysterical symptoms, when recalled to the memory after long intervals, can act powerfully and directly in relieving those symptoms, just as under ordinary circumstances a painful occurrence in the past life, when called to remembrance may excite a flow of tears.

II. It is strange that long past events should have so great an effect that the memory of them should not have faded away. In this respect the occurrence or not at the time of onset of an energetic reaction against the disturbing force is of chief importance. By the term reaction is understood the whole group of voluntary and involuntary reflex actions—from tears to acts of vengeance—by which in common experience the emotions discharge themselves. The emotional after-consequences are attenuated in direct proportion to the thoroughness of this reaction. If an injury has to be borne in silence and any show of resentment suppressed, the psychical and physical consequences are more intense. Full discharge of an emotion may be accomplished by deeds or by their substitutes—words; for instance, relief from the burden of a painful secret is obtained by confession or confidential communication. When such outlets are prevented the memory of a painful occurrence is intensified and bound up with distressing emotion. This reaction (Abreagirung) is not the only kind of relief at the disposition of a sound psychical mechanism which has sustained injury. The painful remembrance may enter into complex associations, be corrected by later representations, or annulled by being stored up with memories of experiences of a contrary kind. A remembered mortification suffers correction by the re-adjustment of subsequent events, by sense of personal merit (self-esteem), and in healthy minds disappears through such associations. Painful experiences with their emotional accompaniments may remain fresh and active for an extraordinary time. These impressions are either completely absent from the thoughts of the patient in his normal mental state, or are present in only the most rudimentary form; they correspond to injuries

insufficiently "reacted" against, and may be divided into two groups: (1) in which, from the nature of the loss or injury, reaction is impossible (*e.g.*, death of a beloved relative, social hindrances); (2) in which reaction is impossible on account of the patient's mental state at the time (*e.g.*, paralysing influence of terror, partial hypnosis). Both kinds may and frequently do occur together. These pathogenic re-representations remain fresh, with unimpaired power of affecting the emotions, because they do not undergo the ordinary wearing away of such emotional disturbances through "reaction" and association.

III. Hysterical phenomena being thus regarded as developing out of psychical (moral) shocks the abnormal state of consciousness in which such pathogenic re-representations arise must next be considered, and the fact emphasized that the memory of the essential causal factor is only to be discovered during hypnosis. That division of consciousness which in certain classical cases has been described as "double consciousness," exists in rudimentary form in every hysterical person, and the tendency to this dissociation and to the development of an abnormal state of consciousness is the fundamental phenomenon of the neurosis, and may be briefly entitled the "hypnoid state." These hypnoid conditions correspond with hypnosis in one respect amongst many points of differences, namely, that representations which arise during them are very vivid, but cut off from associative connection with the other contents of consciousness; they are, however, associated with one another, and vary in kind and degree just as hypnosis may vary from somnolence to somnambulism.

Such hypnoid states form the soil in which morbid recollections with their physical consequences grow. The two together make up the hysterical disposition, but a severe injury (traumatic neurosis), painful suppression of feeling (sexual affections), can in healthy persons cause a separation of groups of perceptions and thus produce a psychically-acquired hysteria. These hypnoid conditions may develop out of the day-dreams so common even in healthy people, and for which the feminine occupations give so much opportunity. Like hypnotic suggestions, they are able to affect the bodily health more powerfully than ordinary perceptions.

Similar considerations apply to hysterical attacks. In the stage of delirium in the fully developed attack, there is an hallucinatory reproduction of the image of memory of the chief injury or group of minor injuries. In fits which consist of motor disturbances only, if one succeeds in putting oneself *en rapport*

with the patient whilst in a cataleptic condition, or attack of sleep, or still better if the convulsion can be evoked during hypnosis, then the remembrance of the injury or group of small injuries which lie at the bottom of the attack can be discovered and made clear. These images of memory correspond to those which have been given as the basis of the permanent symptoms of hysteria. Our observations have proved further that such remembrances which have previously provoked fits, become powerless thereto, if during hypnosis a "reaction" against them and an "associative correction" of them can be brought about.

It follows from the foregoing, that in hysteria the groups of perceptions which arise in the hypnoid states are connected with each other, but shut off from other associations, and thus form a rudimentary second consciousness. The permanent symptoms correspond to a disturbance of innervation of the organs formerly under the control of the normal consciousness by the intrusion of this second state, and the hysterical attack, to a still higher organisation of the latter, by which it gains a complete mastery over the whole personality. Charcot has enunciated the doctrine that an hysterical attack forms the rudiment of a secondary state of consciousness. The normal consciousness is not always completely abolished during a fit; it can perceive the motor phenomena of the fit, whilst the psychical occurrences are beyond its ken.

Frequently there is a kind of equilibrium between the different psychical states present in the same person. Fit and normal life alternate with each other without influencing one another. The attack may appear spontaneously, as is the case with normal images of memory, or may be provoked by irritation of hysterogenic zones, or by the similarity of a new experience, to the pathogenic one, just as a particular image of memory can be aroused through the help of its associations. The authors' mode of treatment is now intelligible. It removes the efficacy of the original disturbance which has not been "reacted" upon, by affording through speech an outlet to the pent-up emotion, and by bringing it into "associative correction." The authors think that this method is productive of great therapeutic advantage; they do not profess to cure the disposition to hysteria, but to prevent the return of hypnoid states. In the "stage of production" in acute hysteria it is valueless, but is effective in removing fits or other persistent symptoms.

Prof. Janet [2] remarks that formerly every writer on hysteria had to give an account of a number of definitions of the disease; latterly the tendency has been, from the infinite variety of hysteria,

to shirk a definition of the disease, or to declare it impossible to define. Thus the authors of recent important books on the neurosis, *e.g.*, MM. Babinski, Pitres, and G. de la Tourette, attempt no general definition, but content themselves with enumerating certain characters by which hysterical phenomena can be recognised as such. But although such a method has its advantages, inasmuch as it allows the phenomena in isolated cases to be studied thoroughly without distraction by theories, yet, on the other hand, it has the inconvenience of producing a very precise idea of an isolated character and a very vague one of the disease itself, and prevents agreement between authors who may believe themselves to be writing of like phenomena, and yet on account of the different teaching of various schools, may be in reality describing distinct ones.

No one expects a definition to explain the essence or very nature of the thing defined; it is sufficient if it expresses in one phrase the majority of the known facts. Like all scientific theories such a definition must be provisional since knowledge of facts constantly increases, but it will hold good until replaced by a simpler one embracing not only the old but the new facts.

1. *Hysteria a Disease of Representation.*—Such a definition should put first the dominant character, and group the other facts around it. The ancient definitions selected as essential a physical character as the name hysteria sufficiently indicates; then Brachet[3] made the convulsive attack the predominant symptom. This was better and more comprehensive, but took no account of the inter-paroxystic period. Briquet's book is the meeting place of the physical and moral conceptions of hysteria; according to him, hysteria is a disease of the emotions, and here is the starting point of the psychological theories of hysteria. Charcot first enunciated these precisely, clearing the ground by demonstrating the characters which distinguish hysterical from organic paralysis. He then showed from the origin, characters, course, and treatment of hysteria that it is a moral (psychical) malady, and confirmed this view by making use of hypnotic suggestion. "Under certain circumstances paralysis can be caused by idea. By reason of the clouding over of the *ego* produced in one case by hypnotism, in another by nervous shock, an idea once installed, fixed in the mind and ruling there without control, will further develop and acquire sufficient force for objective realisation." Here we have the conception of the influence of suggestion and of "fixed ideas." M. Georges Guinon further elaborated the idea of suggestion, and Dr. Möbius generalised it. He writes: "One

view tends to become established, namely, that hysteria is a psychosis, and its characteristic is a morbid mental state." This mental state manifests itself by bodily symptoms. Hysteria differs, however, from insanity, in that in the latter the actions of the insane person are only indirectly in relation with his delusions, the insane person bestirs himself in obedience to an imaginary order, but in hysteria the relation is more direct, thought is transformed into movement without the mediation of the subject's will. Strümpell, expressing analogous ideas, writes: "Certain over potent representations, certain associations of ideas too easily become the point of departure for a long series of apparently physical accidents." He explains these neuropathic phenomena as dependent on idea (Vorstellungen).

The new definition is then that *hysteria is an aggregation of disorders depending on representation.*

2. *Double Consciousness.* — A great number of hysterical phenomena do depend on fixed ideas, upon suggestion, but they cannot all be thus accounted for. (1) There are two kinds of hysterical ties or spasms: (*a*) those which take place when the patient thinks of them, and disappear during sleep, or when his attention is diverted; (*b*) those which persist in spite of all distractions, and sometimes in spite of sleep. These latter must long ago have passed out of the domain of consciousness. (2) Convulsive attacks, which recur with monotonous regularity, seem to depend on physical causes rather than on any thought or idea of the patient. (3) The more direct mental phenomena, which belong to hysteria, such as delirium and somnambulism, are connected with a fixed idea. In short, putting aside the "stigmata," it is impossible to regard the paroxysmal phenomena (accidents) as due to physical changes produced by representations in consciousness. To change the point of view, we will take "the somnambulistic state" as the main point of the definition. This state in every variety of it has been shown to occur in hysteria; lasting sometimes for weeks or months, and giving rise to "double personality." The only constant and essential character of somnambulism is the forgetfulness of all that has passed in the somnambulistic, when the patient has returned to the normal state. Generally the lost remembrances are regained when the subject passes again into somnambulism, and he may thus live two lives, or have two psychological existences alternately succeeding each other. Somnambulism, from the simplest to the most fully developed form, is always the result and the manifestation of a double personality. Somnambulism can be provoked by various

causes, which, however, do not create or form the double personality, but evoke it when it is already existent. Using somnambulism in this sense, many hysterical phenomena, *e.g.*, periods of double existence or automatic action, states of ecstasy, of catalepsy, of delirium, may be looked upon as specially modified forms of it. Further, M. Charcot has shown that close relations exist between the somnambulistic state and hysterical convulsive attacks; symptoms present in the one are also present in the other; they are both amenable to the same influences, the subjects pass readily from the one state to the other, and memory of them is also reciprocal; the patient remembers in the convulsive attack the features of the somnambulistic state and *vice versâ*. Most often the convulsion is excited by an emotion, dream, or fixed idea which is a part of the second (somnambulistic) existence. A striking feature about hysterical convulsions is that they always take precisely the same form in the same patient; for one individual the events are always the same even during a period of years. Similarly for the somnambulistic state, the sufferer always performs exactly the same actions, with the same automatic regularity. The second existence is often a rudimentary psychologic existence, in which a few sensations and ideas exist, not controlled nor modified the one by the other. The fixed ideas which play so large a part in the waking state in hysteria are still more powerful and develop themselves with greater regularity in the second existence. Examining more closely the features of the convulsive attacks, some of them are obviously reproductions of an event in the patient's life; others, such as simple convulsions, cries, &c., are to be regarded as expressions of the emotions. By throwing the patient into the somnambulistic state, and profiting by the memory he has of the phenomena of the attack whilst he is in this state and only whilst he is in it, it is found that some emotion, as terror, anger, despair, always initiates the attacks. All hysterical attacks are of the same nature, and consist in a more or less complete reproduction in a second existence of a past idea, adventure, or emotion, that is, of a more or less rudimentary somnambulistic state.

The above considerations apply to an important category of hysterical troubles, namely, the paroxysmal manifestations, but fail to account for the permanent symptoms. The author has tried to unite both in one explanation by the study of the manifestations of the second personality in the intervals between the somnambulistic states and between the convulsions. The study of the influence of suggestion in producing post-hypnotic actions

first suggested this explanation. The subject has been told to do something in eight days' time, and without any memory of the suggestion, without calculation or reflection, he does it at the appointed time. Instances of the same kind are the spontaneous unconscious actions and automatic writing of mediums. There is thus a second mind existing behind or below the normal one. The facts may be summed up thus:—"These apparently sub-conscious acts are not distinct from somnambulism, but make up a somnambulistic state itself, no longer isolated, alternating with wakefulness, but prolonged without interruption through the waking state. A double personality results, because all psychical phenomena are no longer united in one and the same personal consciousness, but in part remain independent under the form of sensation or elementary images, and are more or less completely aggregated to a new personality, independent of the normal (original) one."

This explains the patient's frequent ignorance of the idea which has provoked the hysterical accident. It is present in the second personality, and its existence can be demonstrated during hypnosis, or as the author has often done by means of automatic writing, but in his ordinary state the patient will be unconscious of it.

Thus hysteria is in a sense a disease of *dissociation* of ideas, and this dissociation plays in it a *role* as important as that of association in normal psychology. "The essential character of this 'disease of disaggregation' is the formation in the mind of two groups of phenomena, the one constituting the ordinary personality, the other, capable of further sub-division, forms an abnormal one, different from the first, and ignored by it." M. Janet observes on this new conception of hysteria that the incomplete state of the normal personality constitutes the hysterical vice and permits the disorderly action of the second personality, *i.e.*, hysterical accidents. The second personality hidden under the first, stronger by so much as the latter is enfeebled, replaces it at the first opportunity and becomes apparent. Breuer and Freud, in the preceding paper, remark that "This division of consciousness that has been clearly demonstrated in some celebrated cases of double consciousness exists in a rudimentary form in every hysterical person, and the disposition to this dissociation, and at the same time to the formation of abnormal states of consciousness—hypnoid states—constitutes the fundamental phenomenon of the neurosis."

3.—*Restriction of the Sphere of Consciousness.*—The above

considerations have a general application to the majority of hysterical accidents, but they leave the permanent stigmata of the neurosis almost entirely unaccounted for. These are an essential part of it, and consist mostly of troubles of sensation, memory, and volition, shown in modifications of movement and of character. Some authors have applied the same explanation to these permanent symptoms as to the paroxysmal ones, and accounted for them on the hypothesis of a fixed idea. For the following reasons the author cannot accept this explanation :—(1) There is a precise event at the onset of a paroxysm due to a fixed idea, but there is nothing in the history of the patients to suggest to them left hemianæsthesia or contraction of the visual field. (2) The former are recognised by the patient, whilst they are often ignorant of the presence of the permanent stigmata. (3) Neither in the somnambulistic state, nor by any method of procedure can the existence of a fixed idea relating to the stigmata be shown. (4) The stigmata have characters of which the patient has no conception, whilst the conception of the accident determines the nature of the symptoms. (5) Fixed ideas are of the most variable character, the permanent hysterical stigmata are constant, and have been the same since the middle ages. (6) The tendency to the formation of fixed ideas, the amenability to suggestion, only takes place late in the course of the disease.

These stigmata take the form of enfeeblement or suppression of sensation, memory, or movements. In the case of anæsthesias the sensory loss is not a localised one, but rather a particular manifestation of an enfeeblement of all the functions of the cerebral cortex. M. Janet considers from various reasons that the stigmata are not due to local lesions of sensory terminations, muscles, nerves, or centres. A state of irritable weakness has been admitted by most authors, but a more precise term is desirable. This cerebral weakness is found chiefly in a lack of the power of attention. Want of volition, aprosexia, hesitation and doubt are the essential psychical characters of hysteria. The weakness of attention is so great as not only to prevent intellectual work, but to modify also the normal life and ordinary thought. An hysterical woman, for instance, is incapable of perceiving several sensations at one time. Her attention is easily distracted, as we say. The "field of consciousness" in hysteria is very small, on account of this readiness to distraction, and may often be fully occupied by a single sensation, understanding by "field of consciousness" the largest number of simple or rela-

tively simple phenomena which can be perceived at any one time. This restriction of the field of consciousness is only one manifestation of the feebleness of cerebration, which may be described as a "special moral feebleness," consisting in the impotence of the subject affected to bring together and condense his psychical phenomena, and to assimilate them to his personality. In this way are explained the traits of character present in hysterical persons, their instability, and excitability, their sudden impulses, passing enthusiasms and whims, their depths of joy and despair, and also such permanent stigmata as the anæsthesia. Professor Janet sums up as follows: "That apparently the elementary pyschical phenomena, are as real and as numerous as in normal persons, but cannot on account of the restriction of the field of consciousness, on account of feebleness of the synthetic faculty, be brought together into one single perception."

The doubling of the personality, brought forward above, does not stand in contradiction to, but is rather the immediate consequence of this feebleness of psychologic synthesis. The facts of somnambulism and of sub-conscious states are to be regarded as secondary groupings of neglected pyschologic phenomena; "as if the system of pyschical phenomena which constitutes personal perception in everybody, was in these persons split up, and gave rise to two or more simultaneous or succeeding groups, for the most part incomplete and separating the one from the other sensations, images and the consequent movements which should normally be brought together into one and the same consciousness." Several authors have accepted this view, and M. Pick, for instance, considers "the diminution of the power of attention, the restriction of (personal) perception, and of motive impulse, as the characteristics of hysteria."

4. *Hysteria a Mental Disorder.*—The tendency of modern writers is to regard hysteria as a mental disease (*e.g.*, *Donkin, Art.*, "*Hysteria*;" *Charcot and Marie, Art.*, "*Hystero-Epilepsy*" *in* "*Dict. of Psychological Medicine*"). M. Janet fully agrees with this view, but points out that a definition must (*a*) include all the symptoms of hysteria; (*b*) characterise hysteria only and distinguish it from all other diseases. (*a*) Now the psychical aspect of certain symptoms is not at first sight obvious; there are (1) the *visceral accidents* of the neurosis; (2) the vasomotor and secretory troubles; and (3) trophic disorders. Under (1) come palpitations, coughs, hiccoughs, respiratory spasms, numerous digestive troubles, and ischuria; under (2) hæmorrhages vicarious of men-

struation, ischæmia and other vasomotor affections of the skin, with anidrosis or hyperidrosis; and under (3) muscular atrophy, slow growth of the nails in an anæsthetic limb, &c. Most of these apparently physical disorders are, however, amenable to suggestion and to various psychic influences. The persistent emotions of the hysterical are a common ground for physiological and psychical phenomena, and a more perfect knowledge of the psychology of the emotions than we have at present may give another aspect to phenomena now apparently purely physical. Other symptoms must remain unexplained, but if hysteria is a mental disorder the mind cannot be affected without other organs being disturbed, and to these disturbances, secondary and subsidiary to the mental trouble, we must attribute many of the symptoms.

(*b*) Is this conception of hysteria sufficiently precise to distinguish the disorder from other mental disorders? In answering this question the limits of hysteria must be defined. M. Janet is disposed to include under hysteria somnambulism—he speaks of true somnambulism and not of hypnotism; for it is found only in hysterical persons, its symptoms correspond to those of hysteria, and it presents the phenomena of the double personality so characteristic, according to him, of the mental state of the latter. He would also include certain mental states, the results of intoxication from alcohol or hashish, and this leads him to add to his definition of hysteria a certain duration or persistency of double consciousness. Further, he points out the resemblances to hysteria of the symptoms in patients on the confines of lunacy, affections grouped together in France under the name of "délire des dégénérés," in Germany, of "neurasthenia délirante." He thinks a number of these patients belong both to hysteria and to this form of mental unsoundness; there is one common striking character, that is, defect of volition. The mode of dissolution of the mind is different in the "délire des dégénérés" the development of another personality is felt by the patient who speaks of it as "possession"; in hysteria, the new element of a double personality is ignored.

To sum up: "Hysteria is a mental disorder belonging to the great group of degenerations, its physical symptoms are rather vague and consist principally in a general lowering of nutrition; it is especially characterised by mental symptoms, of which the principal is enfeeblement of the faculty of psychic combination (synthesis), a restriction of the field of consciousness; a certain

number of elementary phenomena, sensations and images cease to be perceived, they constitute the stigmata; there results a tendency to complete and permanent division of the ego (*personalité*), to the formation of several groups of mutually independent phenomena; these systems of psychical events either alternate or co-exist, giving rise to the convulsive attacks, somnambulistic states, and sub-conscious acts; finally, this defect of synthesis favours the formation of certain parasitic ideas which, under cover of the control of the personal consciousness are developed independently, and show their presence by disorders of the most variable kind, apparently physical in nature, *i.e.*, the accidents or paroxysmal symptoms."

To put the above in two words, "Hysteria is a form of mental dissolution, characterised by tendency to permanent and complete doubling of the personality."

Professor Janet[4] in lectures at the Saltpêtrière makes a further contribution to the psychological side of hysteria; he concludes from his observations on patients that hysterical anæsthesia is not an organic disease, but a mental, a psychical disorder. It exists neither in the limbs nor in the spinal cord, but in the mind represented—if you will—in the cerebral cortex. In the mind one special phenomenon is concerned; hysterical anæsthesia does not consist of an alteration of the elementary sensations which remain unaffected and retain all their properties, but concerns the special operation of personal perception in connecting new sensations one with another and bringing them into relation with the ego; it is due to a failure of this synthesis of psychical elements that the author has elsewhere named (see last paper), psychical or mental disaggregation. Hysterical anæsthesia is thus a disease of the personality (ego).

After having first demonstrated the frequency of amnesia (loss of memory) in hysteria, Prof. Janet points out the analogies between this loss of memory and hysterical anæsthesia. Just as in the case of anæsthesia so in hysterical loss of memory there is no destruction of the elementary mental phenomena, sensations and images; there is merely an impotence, an insufficiency of the central power (highest centres). There is always a perception by the ego, but one incapable of welding the elementary phenomena into due relation with the personality as a whole. At one time presentations (images) are neglected at hazard, and vague continuous losses of memory are produced; at another

certain presentations having definite characters seem to pass unperceived, and the most curious "localised" losses of memory occur. He believes that these localised losses of memory are determined by the anæsthesia present at the time, or, rather, by the variations in the affection of consciousness by sensations. Very often more complex influences are at work, and this explanation only applies to particular cases. In the third lecture the writer deals with the abnormal susceptibility of hysterical persons to suggestion, that is, to suggestion in its pathological form as a symptom of psychical disorder. He has tried to show that this symptom does not only depend on the laws of association, such as are applicable to all men, but on a peculiar infirmity of the will. This want of will power is present in several mental diseases, and in particular forms the essential symptom of hysteria; it does not consist in the abolition of all voluntary acts in themselves, but is of the same nature as hysterical amnesia and anæsthesia.

Both Dr. P. Blocq[5] and Prof. Grasset[6] deal with Prof. P. Janet's writings on the pyschological origin of hysteria. Dr. Blocq accepts Prof. Janet's theories, and applies them to the explanation of the various phenomena of hysteria. Amongst the permanent "stigmata" of the disorder, he classes on the intellectual side, amnesia and defect of will power. To the so-called condition of "suggestibility" he attaches important consequences. Normally, when an idea is called up, it is utilised in a particular manner, and of its diverse connections only one is voluntarily selected, and the other associations are neglected. The weakening of voluntary control in the hysterical interferes with this process of selection, and a succession of associated images arise, which result in hallucinations and in various actions.

Prof. Grasset, after showing how far Prof. Janet's views elucidate hysterical phenomena, criticises them, and especially his concluding definitions of the neurosis as follows. He says that Prof. Janet's analysis of hysterical psychical phenomena, ingenious and true as it is in the main, demonstrates that this neurosis is a malady of cerebral or more exactly of cortico-cerebral origin. The dissociation of the sub-conscious from the conscious is a pathological action of the cerebral cortex. The conclusion is that hysteria is a neurosis *principally* of cortica origin. Prof. Grasset says principally because he does not regard it as proved that the above-described psychical characters

are absolutely constant and universal, but believes that hysteria is a neurosis of the entire nervous system, in which the brain bears the chief part. Hysteria is only a mental affection, if every disease of which cerebral psychic phenomena form a great part is mental, but not otherwise. The hysteric knows that his aberrations are pathological, but the really insane person believes his diseased state normal, and considers his delusions realities. The centres below the level of consciousness may be pathologically dissociated from those concerned in consciousness, without consciousness itself being changed. A further degree of disturbance concerns the latter as well.

Hysteria can only be properly considered a disease of the personality if both kinds of centres, those concerned in, and those below the level of consciousness, are included under this term; if the two are separated it cannot be so described. It is better therefore to continue to use the term mental in the old and generally accepted sense, and to say that recent researches have demonstrated the psychologic origin or cerebro-cortical formula of hysteria, and not that they have shown it to be a mental disease.

Dr. Blocq,[7] in another paper, considers the theory of Prof. Janet that hysterical anæsthesia is a dissociation of re-presentations too exclusively subjective. He explains in the following way (see also a paper by Onanoff on "Unconscious Perception"[8]) the mechanism of perception of sensations and those modifications of it found in hysterical anæsthesia. The afferent impulses which leave sensory surfaces pass to lower reflex centres, and after a first elaboration there to higher centres, where they are brought into complex associations, and a process of selection then takes place which forms the stage of perception. This final synthesis or selective perception ends in the setting in action of a motor centre, movement occurs and the perception is called conscious (apperception) or unconscious according as the sensory impressions consecutive to the movement return or not to the associated centres and are there identified. In hysterical anæsthesia this series is incomplete. The afferent impulse from without passes unperceived because the action of the associated centres is disturbed; some of them enter into fixed combinations with other centres, which fresh afferent impulses are unable to disturb, and thus the process which ends in identification and in conscious action does not take place. This fixed morbid associated action of nerve-centres, which prevents the proper reception of sensory stimuli, may, however, be broken up

by a weak psychical agency, such as suggestion, and hence the instability of hysterical anæsthesia. One psychical characteristic of hysterical anæsthesia, which is common to all varieties, and not capable of simulation, is a shortening of the reaction time; thus, in hysterical hemianæsthesia the reaction time is always shorter on the anæsthetic side. This is a valuable objective sign.

Mental State and Character in Hysteria. — Dr. Löwenfeld observes that the traits of character which have been ascribed to hysteria by most writers until recently, are the exception and not the rule; when they exist they are due to the coincidence with hysteria of an inherited mental taint (degeneration). When the mental characteristics of hysteria are spoken of, it should be remembered that six times as many women as men are affected with hysteria, and that the distinguishing features of the female mind as compared with the male consist of a weaker will, and greater predominance of the emotional over the purely intellectual faculties. Hysterical persons often fulfil their duties well as daughters, wives, and mothers, and are not always seeking to attract attention to themselves and their complaints.

He goes on to say that Blocq and G. de la Tourette attribute the variations in the psychical condition of hysterical patients to an increased "suggestibility," and regard this as characteristic of the hysterical mental state. But this is not present in every case, and is not or only in small part an independent primary symptom. In cases of "nervous shock" (traumatic hysteria), indeed, such a state of increased suggestibility (readiness to react to suggestion) similar to that produced by hypnosis does occur. It is conceivable that when such a condition is present it may soon disappear, whilst its consequences, in the form of paralysis or arthralgia may persist. No further hysterical symptoms arise, and this limitation of symptoms, in a number of cases, has led to the conception of a "local" hysteria. Breuer and Freud describe as the basis of hysterical phenomena the occurrence of hypnoid states. If the enquiry be not restricted to a state of suggestibility, but the psychical condition considered as a whole, it is obvious that hysteria has a basis to work upon which varies with the social position, education, favourable or unfavourable circumstances of the patients. Their surroundings must have an important influence on the psychical manifestations, and one must not attribute to the neurosis, symptoms which have in reality an independent origin. The character, nature, and fre-

quency of the hysterical phenomena have a great influence on the accompanying mental state.

Memory.—Many disturbances of memory occur in hysteria which we do not find in neurasthenia, at least in the same perfection. The power of reproduction in memory of past experiences or of acquired knowledge may be impaired in neurasthenia, but the defect concerns all remembrances in the same proportion, and complete gaps for certain groups of events, or things learnt are not found. On the other hand in hysteria such gaps in the memory are not uncommon, and affect generally short, but occasionally long periods, weeks or months of the past life. Thus one patient, paraplegic after an hysterical fit, lost all memory of the preceding three months, and another, who spoke English fluently, forgot it completely as her hysterical symptoms increased. Many patients fill in the gaps in their memory with fabrications, and thus their statements vary as to trivial occurrences, or if they keep to the same story it does not correspond with the actual facts. In the majority of cases in which hysterical patients make false or exaggerated statements, the defective memory must be borne in mind, as a tendency to lie is not a symptom of the neurosis.

Hysterical suggestibility presupposes an increased "auto-suggestibility" (increased readiness to react to intrinsic stimuli). After mental shocks, for instance, an abnormal state of consciousness occurs in which there is increased reaction to suggestions, and this favours the production by auto-suggestion of various physical troubles. This occurs frequently in children who are liable to hysteria, in an epidemic form. The author agrees with Gilles de la Tourette in attaching importance to dreams as a source of auto-suggestion. We may use this increased suggestibility as a means of cure in various ways.

Emotions. Suicide.—Increase of emotional excitability varies much in individual cases, shows no correspondence with the severity of other hysterical symptoms present, and is probably due to the patient's original character and training. With this increased emotional excitablity is connected the tendency to suicide, a real danger in hysteria, and one not to be under estimated. During somnambulistic attacks attempts at suicide may be made, and the tendency to it is especially obstinate in the complication of hysteria with melancholia. Lastly, the suicidal impulse may be of uncontrollable character, and independent of melancholic ideas. Another condition to be mentioned is the restriction of the sphere of interest or of feeling, perhaps associated with intense absorption in an unworthy object.

Dr. Möbius[9] quotes Oppenheim as follows: "The fundamental phenomenon of hysteria is irritable weakness, abnormal irritability and readiness to fatigue. Extrinsic and intrinsic stimuli have a greater effect than in the healthy. The morbific factor is chiefly the greater readiness with which the emotions can act on motor, vasomotor, sensory, and secretory mechanisms. Expressed physically, the molecular movements take place with less internal friction. Sensory impulses cause more powerful stimulation and more easy discharge of centres. Hysterical paralysis exhaustion arising from the relatively over-powerful stimuli; it is a true paralysis resulting from resistance to conduction at the point where the will grasps the motor centres, offering more or less considerable resistance to voluntary movement. Although the individual cannot carry out the desired movement, this can still be accomplished reflexly, or as a part of an associated action. A similar condition to that of hysterical paralysis is present in the healthy when the stimulus to action is not only relatively, but absolutely excessive to the "Labilität" of the nervous system (*e.g.*, "traumatic neurosis").

So far Dr. Möbius finds no essential difference between his views and those of Oppenheim. He thinks that the physical changes in hysteria are brought about by idea. Even in health lively sensations of pleasure or pain may cause physical disturbance (organic change), and in hysteria similar changes may be produced with unusual ease, whilst besides these others may occur which are not generally found in the healthy. The nature of the idea in the above conditions is not important as regards the nature of the disturbance, but in ideas artificially excited (suggestion), the character of the subsequent results can be foretold. Analogous states to suggestion occur in ordinary life, and whether dependent on emotion or suggestion there is a connection of cause and effect between idea and physical changes.

Oppenheim, however, in opposition to the above theory, which has, at any rate, the merit of clearness, goes on to argue that hysterical symptoms arise (1) through emotions; (2) reflexly. With regard to (1) his views tally with Dr. Möbius's, for an emotion is nothing more than an idea associated with lively feelings of pain or pleasure, but he errs in failing to recognise suggestion as a cause of hysterical symptoms. Very many hysterical symptoms, and the recognition of this fact is due to Charcot, are, however, unintelligible unless we compare the contents of the idea with the nature of the symptoms. Oppenheim makes a retrograde step in accentuating the importance of reflex action as a cause of

hysterical phenomena; formerly, everything that could not be readily explained was called reflex. Recent advance in knowledge is to be attributed to the recognition of the fact that the nervous processes underlying hysterical phenomena are carried on in the highest parts of the brain, in those whose action is bound up with consciousness. Unless the conception of reflex actions is unduly stretched, they cannot be called in to explain hysterical symptoms.

Every event in the world is due to change of movement in material particles. It is known, however, of some few nervous processes that the material or extrinsic side is only a part of the matter, and that some motions in the brain arise from within, or are intrinsic (will and idea). Every deeper conception of the universe recognises that this is not only true of the brain, but that all material motion is but the extrinsic expression of will and idea. If this be so every process whatever can be considered from a two-fold aspect, an extrinsic and an intrinsic one. In most things we are practically limited to the extrinsic, the intrinsic point of view being confined to our own individual internal consciousness. After long effort the identity (? concomitance) of conscious movements with material changes in the corresponding nerve centres has been established by inductive reasoning. The next step is the recognition of a corresponding inner psychical side, not only to conscious cerebral processes, but also to those which are considered as reflex. Observation fails to indicate any essential difference between psychical reaction and reflex action. Whether more or less is interposed between the afferent and efferent limbs of the reflex arc does not alter the nature of the process. The only difference is that the inner aspect of the process sometimes falls within our consciousness and sometimes does not. Every psychical process is thus a reflex action, and every reflex action a psychical process. If a distinction is sought for, then the expression reflex action must be restricted to those nervous reactions, whose intrinsic aspect remains inaccessible to cerebral consciousness as is the case in the spinal reflexes. In this sense reflex action plays but a very subordinate part in the pathogenesis of hysteria. The tendency of workers in natural science, which in general deals only with the extrinsic point of view, to fix exactly the manner of being from this aspect of that which can only be known from the intrinsic, leads to confusion and unsound hypothesis, because of the scanty knowledge of the motions of matter in the brain. Hence the inadequacy of anatomico-physiological explanations in hysteria and the erroneous explanation of hysterical paralysis by obstacles to conduction of nervous impulses.

Lastly, Dr. Möbius objects to Oppenheim's separation of the traumatic neurosis from hysteria. He thinks that the name should be retained on the ground of practical convenience, but that the traumatic neurosis is a form of hysteria differing only in degree and not in kind.

According to Dr. Cousot,[10] hysteria may show itself by an isolated symptom such as monoplegia or contracture, and this form is especially common in children. Charcot insisted that the face is never involved in hysterical paralysis, but several cases of this kind have now been observed. Hysterical anæsthesia is held to be a disorder of the personality, and the author tries to support this hypothesis by a reference to the events of every-day life. If we set about a mechanical task we go on with it unconsciously, even if a train of thought altogether foreign to it suddenly starts up and absorbs all our attention. Whilst our whole attention is fully concentrated on some fixed purpose, other events which take place in the neighbourhood may, although they pass unperceived by us, be registered in our brain. So in hysterical anæsthesia, sensory impulses passing to the brain can set free nutritive and secretory reflexes and yet not rise into consciousness. Study of hyperæthesia teaches us the meaning of many otherwise unintelligible actions of hysterical patients. Just as a patient with hyperæsthetic retina shuns the light, so hyperæsthesia of the gastric mucous membrane causes anorexia and obstinate vomiting. The onset of pains in the head, with vomiting and fever, may simulate the symptoms of meningitis, and violent pains in the abdomen with vomiting and constipation resemble those of peritonitis. Similarly the symptoms of angina pectoris and of arthralgias may occur from hyperæsthesia of the bones and joints. Dr. Cousot relates a case in which anorexia, vomiting of blood, anuria, rapid respiration (30 to the minute during a period of four weeks), successively occurred; the dyspnœa ceased during sleep. The motor disturbances in the form of paralysis, contracture or tremor, so frequently present in hysteria are attributable to the weak condition of the muscles which is indicated by the dynamometer. The soil is prepared through deficient innervation from the nervous centres; then an injury, a psychical shock or an hallucination makes the latent paralysis manifest. The tendency to contracture is an indication of the increased irritability of the pyramidal tracts, and tremor is also a consequence of this. Although hysteria is clearly a functional neurosis, yet it may affect the trophic centres. Babinski, in 1886, described

muscular atrophy as present in some hysterical cases, and the author has seen a case in which muscular atrophy occurred in the region of distribution of both popliteal nerves with hysterical pains in the joints; examination under an anæsthetic showed that the joints were healthy and there was no reaction of degeneration in the muscles; the case was one of atrophic paralysis of hysterical origin. Tabes, syringomyelia, and especially paraplegia, are closely simulated by hysteria, and the diagnosis, whether the symptoms are due to organic or hysterical changes, is by no means always easy. The author concludes that the true nature of this many-sided neurosis is more likely to be reached if its explanation be sought for on the basis of known physiological laws and hysterical patients not simply dismissed as past masters in lying and dissimulation.

Experimental physiology has shown that prolonged intense or repeated stimulation of a motor centre may spread to other centres and give rise to general convulsions. Excitation of other non-motor centres may be similarly disseminated through the brain, and thus an emotion affecting first a purely psychical centre may stimulate sensory and motor ones and react on the organic functions. An emotional stimulus, by its dissemination through psychical and then through sensory and motor centres, gives rise to the series of hysterical symptoms. It is diffused in this manner because the central nervous system is over-irritable, is easily affected by suggestions of diverse kinds; this suggestibility characterising the hysterical mental state. Hence the unconscious production of a whole train of morbid phenomena, best seen and most thoroughly studied in traumatic hysteria.

II.—Papers on Miscellaneous Facts concerning, and Symptoms of, Hysteria.

Etiology of Hysteria and Neurasthenia.—Whilst all allow the influence of heredity in the causation of hysteria and neurasthenia, there is, as Dr. Löwenfeld observes, divergence of opinion as to the part played by it. At one extreme are Arndt, Charcot and the school who hold, the former for neurasthenia, and the latter for hysteria, that heredity is the only predisposing cause, all others being exciting agents (*agents provocateurs*). Arndt thinks that in neurasthenia there is a failure in the full development of the nervous system, either innate or acquired, so that the persons affected remain undeveloped or childlike in character. With this imperfect development a chlorotic blood-state is commonly

present. The origin of this nervous-chlorotic constitution is derived from an inherited weakness in the progenitors, and is an expression of degeneration. The large majority of German authors see in the inherited neuropathic constitution a predisposing factor to neurasthenia which is frequently present, but is not constant, nor the only one. According to Levillain, there is an hereditary form of neurasthenia, the inherited neuropathic taint manifesting itself in certain symptoms which complicate those proper to this neurosis. The author found that seventy-five out of 100 cases of neurasthenia under his care, gave evidence of inherited nervous predisposition; in twenty-nine the mother, in six the father, and in seven cases both parents suffered from "nervousness," neurasthenia, or hysterical attacks; and in the remaining cases from other nervous disorders. He has found hereditary tendencies present in a large proportion of hysterical patients, but not in all, and that the worst forms of hysteria may be developed without any such influence. The disposition to hysteria which the brain in one case has acquired through inheritance, it acquires after birth in another through disease, such as meningitis, or through the persistent action of harmful agencies on the nervous system. How can epidemic outbreaks of hysterical symptoms, which sometimes occur amongst the people of a whole district in whom no inherited tendencies to nervous disorders have before been noted be accounted for, if the disposition to hysteria cannot be acquired? He agrees, with other writers, that in the great majority of cases of hysteria the heredity when present is a direct one. The action of the hereditary tendencies varies in particular cases remarkably. On the one hand, the children of an hysterical mother, brought up with the utmost care, and shielded from all harmful influences, early show signs of hysteria for which no exciting cause can be discovered, and on the other, patients similarly predisposed to the neurosis preserve their good health for a long time, or at the most, suffer only from certain nervous symptoms until a powerful shock or persistent depressing influence evokes hysterical symptoms.

The so-called Signs of Degeneration.—Dr. Löwenfeld thinks that too much importance must not be attached to the minor malformations, chiefly of the special sense organs and cranium, which have been described as signs of degeneration. Severe cerebral damage from hereditary taint is often shown by clear signs of bodily degeneration, but there is no constant relation between the bodily and mental anomalies. In many thus hereditarily

affected in spite of evident signs of mental degeneration those of physical degeneration are absent, and *vice versâ*. According to Magnan the characteristic features of hereditary degenerations lie in the mental state. There are the cases of agoraphobia, claustrophobia, aichmophobia, pyrophobia, folie de doute, délire du toucher, and all the group of uncontrollable impulses. Koch divides the psychopathic conditions (states of lowered capability) into three kinds; the psychopathic disposition, the psychopathic taint, and psychopathic degeneration. In hysteria and neurasthenia, the psychical changes belong to the two first or less serious conditions; but it must be borne in mind that anomalous mental states are found in neurasthenia apart from any hereditary tendencies. Arndt's view that the nature of the change in neurasthenia is essentially a want of full development of the nervous system has been mentioned, but such an arrest of development in the hereditary form of the disorder has not been made out. The author attaches more importance to variations in the development of the vessels of the brain, and therefore, in its nutrition. He points out that in no organ is the influence of nutrition, or capacity for work so clearly shown, as for instance, in the production of fatigue or exhaustion, or even loss of consciousness by hunger, hardships, over-fatigue, and loss of blood. Variations in the size of the vessels must be of importance, because they affect the blood supply, and no development of the cerebral vessels can be considered normal which does not bear a certain relation to the size of the brain. From his own observations, the author has found that the relation of the width of the arteries to the brain weight, varies very considerably. Physiological and pathological observations show that for the recuperative power or elasticity, which is so important a property of nerve-cells, the condition of nutrient supply is of the greatest importance. Defective development of the vessels of the brain entails a predisposition to all the disorders produced by causes which exhaust the nerve-centres, and interfere with brain-nutrition.

Influence of Sex.—The proportion of men to women affected with hysteria is put at 1 to 17 in Germany, whereas Gilles de la Tourette, in Paris, and Pitres, in Bordeaux, found it to be 1 to 3, so that the conclusion is that in the lower classes from which both series of statistics were drawn the disposition of men to hysteria is much greater in Paris and in Bordeaux than in Germany. In the better classes frequenting health resorts, the author found the proportion of male to female cases to be 1 to 6, and in childhood 1 to 2. The author endorses the commonly received view that

the mental characteristics which distinguish women from men—the predominance of the emotions over the intellect—is the foundation of the greater tendency of women to suffer from hysteria.

As regards neurasthenia, Beard and Bouveret think that it is more frequent in men, Möbius in women, and Levillain that both sexes suffer equally. The author's own observations have been that most men suffer, but he thinks this may be partly accounted for by the fact that so many women with nervous troubles consult the gynæcologist and not the physician.

As to occupation, neurasthenia is especially frequent in those who work with their brains rather than with their hands, particularly if the occupation yields a slender and precarious means of subsistence, with much anxiety, responsibility, or interference with proper rest. Hysteria does not especially affect this class. The author cannot confirm Gilles de la Tourette's statement that hysteria is most frequent in those of the labouring class whose occupation exposes them to accident, and hence to traumatic hysteria; he thinks that workmen who have received injuries to their nervous systems suffer from neurasthenic conditions, and not especially frequently from hysteria.

Race and climate have no especial influence.

Exciting Causes—Psychical Overwork.—Arndt puts the question in another point of view from that generally stated, namely, that neurasthenia is produced in very many cases, not by mental work absolutely excessive in itself, but by a relative incompetence to fulfil the daily task. The author distinguishes three groups of cases in which excessive brain work acts as the cause of neurasthenia and hysteria.

(1) Those persons who have undoubtedly fallen ill by reason of their daily work, although they have not worked extraordinarily hard, but have solely performed the average duties of their place, or even less than these, form a great many of the cases. Examination candidates who break down after a course of study of only average severity afford an example. Again, in our times the ambition of parents for their children to rise out of their own class makes them put the children to unsuitable occupations, and thus many persons who would have remained healthy at mechanical trades, when put to continuous sedentary occupations involving brain-work, break down because they are not fitted for them.

(2) The second group comprises persons who have undoubtedly worked excessively hard, but even in them the nervous exhaustion is not to be entirely attributed to overwork, but is also due

to concomitant emotional disturbances of a depressing character, *e.g.*, hard work, with uncertainty of making a livelihood by it; the being superseded by a junior after years of hard work.

(3) The smallest class consists of those who suffer simply as a consequence of excessive brain-work.

Overpressure at School.—The author does not agree with Charcot's view that it is impossible to overwork school children, because when the limit of their brain power is reached they simply give up, and cannot be urged on to make fresh exertion with a tired brain as their elders can, but, on the contrary, thinks that in children it is just as easy to establish a condition in which by an effort of will the over-fatigued brain permits an attempt at work beyond its powers. Hertel, in Copenhagen, found seven per cent. more invalids amongst the children of a school with the longest hours of work than amongst those who worked for shorter times. Axel Key, in Stockholm, found five per cent. more sickness in the first than in the second class. With regard to the question of how far there may be overpressure in the system of education in the German higher schools (Realschulen, Gymnasien, Töchterschulen), the author says that for half of the higher classes there is certainly possibility of excessive mental strain, and for the other half overpressure may be indirectly occasioned by the lack of sufficient time and opportunity for the care of the body and physical exercise. Neglect of fresh air and exercise is most injurious, the mind being developed at the expense of the body. This want of care for the physical health and excessive stimulation of the brain is especially hurtful, because a large proportion of the children frequenting these schools have inherited a disposition to nervous troubles. Muscular over-exertion seldom plays the part of sole exciting agent of hysteria; mostly it acts in association with emotional disturbance or other injurious influences.

Erb,[11] in a speech "On the Increasing Nervousness of our Times," defines this nervousness as a condition of weakness, exhaustion, and increased irritability or a morbid increase and persistence of the physiological processes of fatigue. Neurasthenia, the most common and important form of this nervousness, consists of increased irritability with great weakness, sense of fatigue and exhaustion on the one hand, and diminished powers of work on the other.

He thinks there has been during the last century an undoubted increase of the sufferers from neuroses, in neurasthenia affecting all classes of workers, especially brain workers, and in hysteria

in the male subject. Any cause which overtaxes, strains and exhausts the nervous system may bring on the diseased state which is to be regarded as a pathological persistence of fatigue (exhaustion). Neurasthenia is a disease of brain workers, hysteria is a result of over-stimulation of the emotions and sensations, but there is no sharp line of demarcation between them. The causes are overwork at school in ill-ventilated class-room, and in preparation for examinations; overwork in occupations arising from the long hours of work necessitated by competition encroaching on the time necessary for rest and recuperation. Many states of nervous weakness result from overstrain on the emotional side, anxiety, cares, fears, disappointed hopes, speculation, religious, social, and political struggles.

The physical causes are muscular overstrain, sexual excesses and aberrations, especially masturbation, abuse of alcohol, tea, coffee and tobacco; severe accidents, especially railway accidents, effects of fevers, influenza and diseases of the blood.

Most important is the nervous disposition (neuropathic) which may be inherited or acquired through any of the causes above mentioned; this forms the basis on which the exciting causes work. Consideration of the causes explains the frequency of neurasthenia in the upper classes, in brain workers, in men more than women, and of hysteria in women rather than in men. The excessive tendency to the neuroses in the Latin peoples, in the Jews, from their mode of life and consanguineous marriages through long centuries, and in the Americans from the life of overstrain they lead, can thus be understood.

Affections of Sexual Organs.—The relations between diseases of the sexual apparatus in women and neurasthenia and hysteria Dr. Löwenfeld sums up under four heads.

(1) The affection of the sexual organs, which may be serious disease, or such disorders as flexions of the uterus, only give opportunity for the occurrence of nervous disturbances. The sexual affection in the majority of these cases acts on the basis of a neuropathic predisposition.

(2) The disease of the sexual organs acts in combination with a number of other injurious influences in inducing nervous disorders.

(3) Sexual affections and nervous troubles arise independently of one another, but are coincident.

(4) The sexual affection is the consequence of nervous disorders primarily arising from other causes, and may then itself lead to an aggravation of the nerve troubles.

In men, diseases and disorders of the sexual organs give occasion for the onset of neurasthenical and hystero-neurasthenical (rarely purely hysterical) symptoms. This seems to occur only in those pre-disposed to neurasthenia. Gonorrhœa and the various inflammatory (acute and chronic) affections of the sexual apparatus which form its sequelæ are the most important diseases in this respect.

With regard to sexual excesses as causes of neurasthenia, the author concurs in the views commonly received.

Other causes of neurasthenia are various forms of dyspepsia enteroptosis of Glénard, possibly nasal affections, errors of accommodation insufficiently corrected, any affection which gives rise to lasting pain. Of organic nervous diseases tabes dorsalis, chronic transverse myelitis, and spastic paraplegia are most often connected with neurasthenia, whilst the association of multiple sclerosis with hysteria is, as has been shown by Buzzard's researches, very frequent.

The acute diseases, such as enteric fever, may be followed by neurasthenia as a consequence of the interference with general nutrition, and of the weakness thus induced; but in the case of influenza, the influence of the specific poison is admitted as directly causing neurasthenia, or hystero-neurasthenical conditions which may last for months or years. Influenza is singular in this respect, as in none of the other acute specific diseases, and the same applies to syphilis, has the author been able to find clear evidence of a direct action of the specific poison of the disease upon the nervous system.

Uric Acid Diathesis.—Ebstein states that no small number of so-called neurasthenics are gouty persons in whom sooner or later the true nature of their disease declares itself. The author thinks that the uric acid diathesis plays an important part amongst the causes of neurasthenia. He also gives a warning against the incautious employment of measures directed for the relief of obesity. In several cases treatment for this end has caused severe neurasthenia.

Symptoms of Hysteria.

Hysterical symptoms are considered by Löwenfeld under two heads: (1) the stigmata; (2) accessory symptoms. The author thinks that the stigmata are relatively more infrequent in South Germany, especially hemianæsthesia, extensive anæsthesia, and hysterogenic zones, both in private patients whose cases cannot always be fully investigated, and in hospital ones.

Hückel is also of the opinion that hemianæsthesia is more common and better marked in France than in England or Germany, and that hysterical paralyses are more rare in Germany than in France. In Tübingen where hysteria is common and not only in mild forms, hemianæsthesia, "ovarie" and hysterogenic zones are rare.

Hysterical Anæsthesia.—As to the origin of hysterical anæsthesia, its greater frequency in some groups of cases than in others shows the importance of the patient's surroundings, *e.g.*, the symptoms presented by his neighbour in hospital. That hysterical anæsthesia is more common in France than in Germany is partly due to unconscious imitation. An hysteric who sees and hears in a hospital that hemianæsthesia is a common symptom in this disease, and further notices that the doctors take a great interest in its occurrence is sooner or later likely to develop the symptom. Other hysterical symptoms, convulsions, mutism, aphasia, so often arise in similar ways that it would be strange if anæsthesia did not also do so. Prolonged physical examination may have a similar influence in bringing out or fixing anæsthesia. Difference of duration of anæsthesia in different patients may partly be accounted for in this way. The action of the so-called æsthesiogenic agents partly depends on suggestion, but nevertheless some obscure physical action of these agents cannot be excluded, for exact imitations of magnets have no effect when true magnets produce an æsthesiogenic influence.

So far Dr. Löwenfeld has not been able to discover hysterogenic zones in the different hysterical neuralgias which he has observed.

Ovarie.—Is the ovary really felt in the "ovarian zone"? The author says that in hysterical and non-hysterical women, with thin abdominal walls, he is certain that in the spot indicated by Charcot, the ovary can be felt, and gives rise to a special feeling of pressure which is not given by surrounding parts. At the same time, he does not mean to say that the painful feelings on pressure always come from the ovary itself; the presence of hyperæsthetic areas in the same situation in men contradicts this.

Hysterogenic Zones. — In discussing Bernheim's view that hysterogenic zones are due to suggestion, and that no influence can be supposed to come from the hysterogenic spots themselves, he states his opinion that, although a great number of the zones found by French authors rest on a basis of suggestion, yet this is not true for all of them, and gives cases in which the hystero-

genic influence was not due to suggestion. The extraordinary readiness with which in the patients in the Salpêtrière, and at Bordeaux, hysterical attacks were excited from the most diverse parts of the body (whether these were anæsthetic or hyperæsthetic) appears to him to be connected with suggestive influence. From a practical point of view these zones have not the importance French authors attribute to them; and in opposition to Pitres, he thinks that they can only very seldom be of therapeutic avail in arresting an attack.

Hæmoptysis in Hysteria.—Hæmorrhage from the air-passages is rare in hysteria, but may be observed in men as well as in women. In the latter, it may be vicarious of menstruation, in which case there is generally little cough. In one male case, reported by Laurent, it never occurred except during an hysterical attack. E. Wagner has met with a sputum of exceptional characters, coming probably from the mouth, in a number of badly-nourished hysterical patients with cough; it lasted weeks or months, and excited suspicion of tuberculosis, although physical examination excluded disease of the lungs and bronchi. The sputum was from 20-100 grammes in quantity in the twenty-four hours, was reddish but not rusty; it contained numerous small grey particles and blood-cells, but no formed elements from the lungs or bronchi.

Circulatory Disturbances.—A feeling of heat in localised areas accompanied by reddening of the skin, and objective rise of temperature of the part, is not uncommon, especially in the face. Brodie observed that in arthralgia first came coldness and pallor, followed after some hours by redness, increased heat, and sweating of the part.

In a patient of Rosenthal's (a girl of 23), discolouration of the hands with feeling of cold preceded hysterical attacks. The temperature of the hands on days free from the attacks, 34·4° C. fell to 30·6° C. before the attack; they became pale, the finger tips and nails blue. Afterwards the temperature rose to 35·6° C. with red fingers and nails.

Hysterical Hæmatemesis.—In hysteria, hæmatemesis may be vicarious of menstruation, but often occurs quite independently of or during the menstrual periods. Certain patients are taken suddenly with hæmatemesis in relatively good health and with almost complete absence of pain. In other cases it comes on amongst other symptoms of the neurosis, in the course of grave hysterical affections. In many patients, burning or gnawing pains, with exquisite tenderness in the epigastrium, occur, and vomiting

of blood is attended with increased pain, anxiety, and sometimes a faint of short duration. In the majority the hæmorrhage recurs, sometimes daily; the quantity of blood in twenty-four hours may amount to a litre, and there may be melæna. Severe anæmia and weakness are thus caused. The author, after the above account, says that the diagnosis from gastric ulcer can only be made with confidence in cases in which hæmatemesis occurs without long preceding gastralgia and dyspepsia, or in which it follows other hysterical hæmorrhages, and that in view of the frequency of ulcer in young women, the presence of an ulcer should be first thought of in diagnosis. The writer thinks that seeing the obscurity of symptoms of gastric ulcer in many cases, too great caution can hardly be exercised in diagnosing hæmatemesis as of hysterical origin.

Hysterical Œdema.—Hysterical œdema, as Sydenham described it, is most marked in the evening, does not pit on pressure, is seldom an isolated symptom, but accompanies paralysis, contracture or arthralgia. It is generally confined to one foot, hand or limb, but may affect more than one limb at the same time. In some cases the skin over the swelling is normal in colour, in others it is cyanotic or even dark blue; Charcot has produced this œdema by suggestion. A lowering of the temperature generally accompanies the œdema. The development and decline is characteristically rapid. The duration depends on that of the motor disturbances accompanying it. Striking changes in the swelling may occur during the menstrual periods or under emotional excitement.

Muscular Atrophy.—It is now known, in opposition to former views, that muscular atrophy may occur in hysteria. Dr. Löwenfeld classifies the cases as follows:—

(1) Simple atrophy.

(*a*) In connection with paralyses and contractures. It may develop very quickly. No fibrillary contractions nor R.D. Electrical irritability falls proportionately to the muscular wasting.

(*b*) Unconnected with paralysis or contracture (isolated affection). Gilles de la Tourette and Dutil say that the atrophy occurs most frequently on the side of the seat of other hysterical symptoms, especially of disturbances of sensation.

(2) Degenerative atrophy, with fibrillary contractions and R.D.

The actual occurrence of muscular atrophy in hysteria is no longer open to doubt. In the diagnosis from atrophies of organic

origin, the degree and extent of atrophy, loss of electrical irritability and presence of fibrillary contractions give no aid. The development of the hysterical form is somewhat characteristic. It comes on remarkably quickly, and reaches a severe degree in a short time. The further course is variable. In many cases it quickly gets better, in others it lasts long, sometimes even for years, stationary. Observations do not yet show whether recovery takes place in all cases, or whether the cases of atrophy with R.D. follow a less favourable course than those of simple atrophy. Of greater weight is the relation of atrophy to other hysterical symptoms. It may almost be said that it exclusively affects parts which are or have been the seat of other hysterical troubles (paralysis, contracture, anæsthesia).

Aphasia.—The author thinks that, in addition to mutism, true aphasia may occur in hysteria, though most cases described as hysterical aphasia are in reality mutism. The author gives a case from his own practice in which motor aphasia followed a severe hysterical convulsion in a girl of 24, and quotes two others, one from Charcot,[12] and another reported by Dobie.[13]

Convulsive Attacks of Hysteria (Löwenfeld).—In answering the question what symptoms are characteristic of the hysterical fit, similar difficulties are presented as in the case of epilepsy. Convulsions are the most striking characteristic of the ordinary epileptic fit, but they occur in very variable degree and may be absent. Paroxysmal loss of consciousness has, therefore, been selected as the essential feature of the fit, but is not an absolute criterion, since epileptic fits may occur without it. In the great majority of hysterical attacks the striking feature is the convulsions, but they may be inconspicuous or absent, and a characteristic must be found which is present in every attack, with or without convulsions. The author selects a certain morbid mental change as this characteristic, and this may vary in intensity from a slight clouding over of perception to the full interruption of the normal stream of consciousness, to the entrance into a psychical state wholly different from the normal (double consciousness), or to apparently complete loss of consciousness. This mental change, however, may be absent in hysteria, just as loss of consciousness may not occur in epilepsy.

The succession of the typical stages of the attack as described by Richet is seldom met with. Single periods of the complete hysterical attack are observed in all their details as at the Salpêtrière, but isolated or joined to one another in altogether irregular ways and in very various degrees of development. This

atypical succession of the different stages of the attack is the most common form. The fully-developed "*grande attaque*" is thus rare in all countries, but variations or modifications of it are often met with, due to the occurrence of one of the periods as an isolated attack. One variety is the isolated occurrence of the epileptoid stage, which may be very difficult to distinguish from true epilepsy. It was formerly believed that hysteria through increasing severity of attacks might later develop into epilepsy, and also that there was a mixed form of attack intermediate between hysteria and epilepsy. But we now know that hysteria and epilepsy are two neuroses, which neither pass into one another nor in any way form a conjoint affection. Nevertheless, hysteria and epilepsy may exist side by side in the same case. Generally, epilepsy is the primary affection. After epilepsy has been long present accidental causes lead to the development of various hysterical symptoms, either of the persistent or of the paroxystic kind.

Hysterical Pyrexia.—In a valuable paper, Dr. Sarbo[14] says that Briquet distinguished three kinds of hysterical fever. (1) Symptoms characterised by the quick pulse, that accompanied them. (2) Quick pulse and subjective feeling of heat without other symptoms. (3) In addition to the above-named symptoms, pains in the head, feeling of thirst, anorexia, faintness, &c.

Bouchut recognised only the form of hysteria with attacks, and distinguished it as a paroxysmal neurosis free from fever, from another neurosis in which fever was present, and which he named "chronic neurasthenia." But this latter is merely a variety of hysteria, and his classification has not been accepted. Under the name of "hysterical fever of pseudo-meningitic type," Boissard,[15] Chantemesse, and others describe a new form. The name of this affection sufficiently describes the symptoms.

Gagey[16] divided hysterical fever into three forms: (1) continuous; (2) remittent; (3) intermittent: the latter may assume a quotidian or tertian type. His opinion was that an hysterical fever exists, of the nature of which we are ignorant, and which often comes on after childbirth, severe illnesses and psychical disturbances; the severity of the symptoms is out of proportion to the elevation of temperature.

Brian[17] recognises three forms of hysterical fever: (1) Chronic, lasting a long time; an irregular febrile condition in which the elevation of temperature does not correspond to the severity of other symptoms; this form corresponds to Gagey's first division. (2) Of short duration, like typhoid, which attends the onset of

hysteria, and in which the temperature reaches 39°-39.5° C. (3) Intermittent, generally assuming the tertian type. He cites several other authors as also describing an intermittent type.

Fabre,[18] who considers hysteria as a disease of the vegetative organs *par excellence*, distinguishes five forms of hysterical fever: (1) ephemeral; (2) chronic; (3) intermittent; (4) typhoid; (5) hysterical febricula. This author also describes cases of hysteria, in which the symptoms predominate in the lungs, and simulate pulmonary phthisis.

Pinard opposes the views of the above writers; he doubts whether there was actually any fever, points out that the amount of urea was not estimated and that no loss of weight is mentioned, which would surely have occurred if there had been fever. Some cases he thinks were abortive typhoid. In others, symptoms were present, resembling those of fever, but in which there was no rise of temperature, and which he calls therefore "hysterical pseudo-fever."

Later, Chauveau distinguished: (1) A slow form, a long lasting condition of moderate fever (38-39° C.) without loss of weight. (2) Intermittent form. Most cases described under this head Chauveau regards as cases of true intermittent fever, but he thinks that hysterical cases also occur. (3) Form of short duration. The pseudo-meningitic type he refers to his second variety. He ascribes the pathogenesis of hysterical fever directly to an irregular action of the heat centre. Debove succeeded by means of suggestion in raising the temperature to 38·5° C.

Of German authors, Wunderlich[19] especially drew attention to the temperature alterations to be observed in hysteria. He emphasises the occurrence in weakly persons, and in nervous persons of hysterical constitution, very often on the slightest provocation, or without any at all, of elevations of temperature which may last a long time. Sometimes the remarkable phenomena of a different temperature on the two sides of the body is noticed. Dr. Sarbo observed several times an elevated temperature on one side of the body, without any organic change in one or other side, in persons suffering from hysteria, or spinal affections.

Rosenthal[20] mentions in severe hysterical conditions local redness of regions of the skin and elevation of temperature with a pulse of 100-130, and an axillary temperature of 37·4-37·6° C. Rosenthal thinks that febrile symptoms may be present in hysteria, but true fever, *i.e.*, an elevation of the body temperature, never. Hysterical fever is, therefore, a misnomer, and leads to confusion.

Hale White[21] gives the chief features of hysterical pyrexia as follows: (1) it always occurs in girls; (2) they are often otherwise hysterical; (3) they are always of that age at which hysteria is most common; (4) ovarian pain and tenderness is often present; (5) rigors may be present; (6) delirium, rapid pulse, flushings, or a trace of albumen in the urine, are often found; but any one or all may be absent; (7) the characteristics of the temperature are its erratic behaviour, both diurnally and generally often varying in different parts of the body, and quite unlike the temperature of any known inflammation or fever, as well as the occasional great height which it may attain.

Moravcsik[22] gives the following summary of his observations: (1) In the hysterical, the body temperature, pulse, and respiration show fluctuations. (2) A rise of temperature occurs without any ascertainable organic changes. (3) The height of the temperature often bears no relation to the rate of the pulse and respiration. (4) The morning temperature is often higher than the evening. (5) The pulse and respiration show the greatest fluctuations. (6) Increased rapidity of pulse and respiration rate often precede a convulsion. Moravcsik also observed an elevation of temperature produced through suggestion during hypnosis; as did also Krafft-Ebing.

A fever, of allied nature, is found in the high temperatures which occur after the status epilepticus. By Charcot and his school, this fever was considered to be due to muscular overwork. Witkowski considers that the rise of temperature is not due to muscular work, but that there is some connection between it and loss of consciousness. He entitles the fever which occurs in general paralysis, uræmia and disseminated sclerosis with loss of consciousness, "sensory fever." He considers hystero-epilepsy as a variety of epilepsy, in which view Dr. Sarbo does not follow him, preferring, with the French school, to regard hystero-epilepsy as distinct from epilepsy, and the fever observed to occur in it as an "hysterical fever."

Its Pathogenesis.—Dr. Sarbo then gives an abstract of all the chief papers, from Virchow's downwards, on the regulation of the temperature by nervous heat centres, and sums up the most important points as follows:—(1) Clinical observations indicate that the central nervous system plays a part in the origin of fever and the discovery of the vasomotor and thermal centres by experimental research confirms this. (2) Those fevers, which have no substratum of organic disease, and are therefore conditioned by the nervous system alone, are to be considered as originating in disturb-

ance of the function of the thermal centres, and may be named "functional nervous fever." (3) It is a *priori* probable that in hysteria, the functional neurosis *par excellence*, such fever should occur.

The author divides cases of hysterical fever into two groups :—

(1) Those described as hysterical fever, but in which no thermometric measurements were taken.

(2) Those in which the temperature was taken comprise two sub-varieties : (*a*) Those in which there was no rise of temperature (chiefly author's own observations). (*b*) Those in which a rise of temperature was present (his own and other reported cases.)

(2a) Hysterical pseudo-fever, hysterical tachycardia. In these cases there are the symptoms of fever, headache, rapid pulse, thirst, dizziness of head, &c., but no rise of temperature. The author draws the following conclusions from six cases of his own and one of Rosenthal's of this nature.

(1) Hysteria as a vasomotor neurosis may show a group of symptoms, which, although tachycardia is associated with them, give the impression of a febrile condition ; since, however, there is no actual rise of temperature, he calls this condition "hysterical pseudo-fever" although he does not include all the cases included by Pinard under this term (Pinard reckoned cases with a temperature of 38-38·5° under this category, considering this temperature physiological).

(2) Hysterical tachycardia is a symptom pretty often observed.

(3) This pseudo-fever appears in simple hysteria as well as in hystero-epilepsy.

(2b) *Cases with actual rise of Temperature.*—The author has endeavoured to make a more satisfactory classification than those hitherto proposed which shall keep in view the pathogenesis of the condition and its analogy with other hysterical phenomena. He divides them into two main classes (1) continued fever ; (2) attacks of fever. Both these varieties can be sub-divided into a severe and mild form according to the height of fever. The continued fever has an analogue in the sensory sphere in the hysterical anæsthesias of long duration, and in the "état de mal" several times observed in hysterical seizures.

Similarly the attacks of fever may be compared with quickly passing anæsthesias, and with single convulsions.

Hysteria { Anomalies of thermal centres (*a*) continued fever (*b*) attacks of fever —severe above 38·0° C.; mild below 38·5°.
" " Sensibility (*a*) persistent anæsthesia (*b*) ephemeral anæsthesia—either may be superficial or deep.
" " Motion (*a*) "état de mal" (*b*) isolated convulsive attacks—either may be severe or mild.

These resemblances are justified by the connections observed in his cases between the convulsive attacks and the febrile states, and in the correspondence from the point of view of the variability of these states and the other hysterical symptoms.

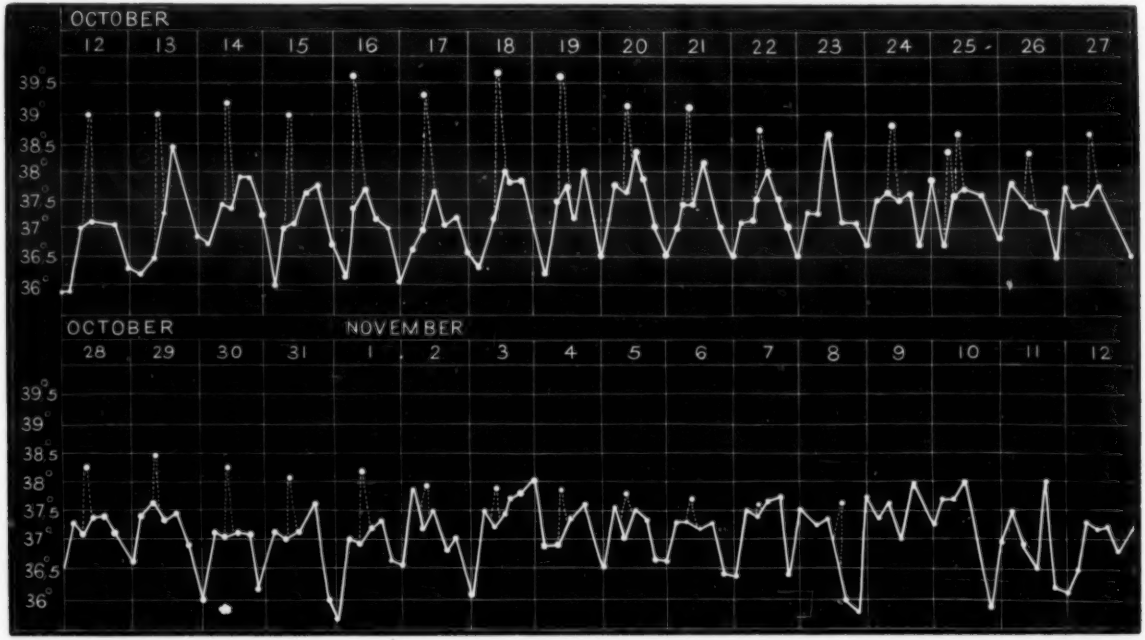
The author cannot agree with Pinard that a temperature of 38°-38·5° C. is physiological; he found it present only exceptionally, in the course of observations taken six times a day for over seven months at a time, and then only in connection with other febrile symptoms, the normal temperature being 37·1°. He considers that this indicates a less degree of the same changes that take place in the heat centres when a higher temperature is produced, as for instance in the case of tuberculosis where a feverish condition is generally spoken of with a temperature of only 38 or 38·5° C.

A. *Hysterical continued Fever.* (1) *Cases with slight rise of temperature.*—Dr. Sarbo discusses some cases at length, and then criticises Charcot's dictum that as a rise of temperature never follows an hysterical whilst it may be present at the close of an epileptic attack, a rise of temperature in any case of convulsions indicates their epileptic nature. This diagnostic aid loses its value, because after an hysterical "état de mal," fever may come on. He has observed a case of undoubted hystero-epilepsy in which, after a severe convulsive attack, a paroxysm of fever came on, and this was not connected with the convulsion, as fever subsequently was present without convulsions. Cases are met with in which hystero-epileptic convulsions are combined with hysterical fever.

In this group are comprised the cases of fever with symptoms resembling meningitis (*fièvre pseudo-méningitique*). He thus sums up the characteristics of the group:—

(1) In the course of hysteria, continued fever, not dependent on organic disease, may appear; it is not to be considered as due to muscular work (in the convulsive attacks), and on account of its dependence on hysteria alone should be called "hysterical continued fever."

(2) Hysterical continued fever can be divided into two grades, according to the elevation of temperature, whether below or above 38·5° C. Its duration varies from days to months.


OCTOBER
12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27
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OCTOBER NOVEMBER
28 29 30 31 1 2 3 4 5 6 7 8 9 10 11 12
39.5° 39° 38.5° 38° 37.5° 37° 36.5° 36°

(3) The fever has no settled type. It may appear and disappear quite suddenly.

(4) Such anomalies as a difference of temperature on the two sides of the body, and a low evening and high morning temperature, are met with.

(5) The severity of the accompanying symptoms often bears no relation to the slight rise of temperature.

(6) The fever may be combined with such a group of symptoms, as to resemble the picture of a known disease, *e.g.*, typhoid, tubercular meningitis, phthisis, and peritonitis. On account of the accidental character of these groups of symptoms, the division of the cases into sub-varieties according to the symptoms present, as French authors have done, is not desirable.

(7) In most cases no cause for the fever is discoverable: in some stoppage of menstruation, psychical shocks, and injuries appear to be causes.

(8) In some cases this hysterical fever comes on after, but is independent of a series of convulsive attacks (ètat de mal hystèrique). Accordingly the onset of high fever is no criterion in the distinction between hysterial and epileptic convulsions.

(9) This form of hysterical fever has so far been observed in the majority of cases of hystero-epilepsy.

B. *Hysterical paroxysmal fever* (attacks of fever), (1) attacks of mild type.

The author reports an instance in a woman of 24 years of age. She suffered from hysterical pains in the abdomen, but neither these nor the retention of urine, constipation, and meteorismus from which she suffered at times were connected with the attacks of fever, in which the temperature rose to 38° C. Fever came on from time to time and lasted for an hour or two, and was attended with accleration of pulse and respiration.

(2) *Attacks of High Fever.*—Case of hystero-epilepsy; the patient, a girl of 16, suffered from severe hystero-epileptic attacks, originally brought on by a fright; from anæsthesia of the right side of the body, right amblyopia with contraction of visual fields. Loss of voluntary movement in the right extremities; the temperature was reduced over the right side of the body. The last attack occurred three weeks before admission, and left a pain in the head. A chart of the temperature is appended. Malaria, typhoid and tuberculosis could all be excluded as causes of the fever, and no organic disease was discovered; the diagnosis of hysterical fever was therefore made by exclusion. The attacks

of neuralgia from which the patient suffered were so changeable in character that they could have had no causal connection with the febrile paroxysms. The rise of temperature came on daily almost at the same hour, with feelings of heat, apathy, somnolence, flushing of skin, which was hot to the touch, rapid pulse and thirst, ended in profuse sweating, and the patient fell into a deep sleep and awoke with headache. The temperature on the right (anæsthetic) side of the body was ·3-·5° lower than on the other side; the author has observed this lowering of temperature on the anæsthetic side in another case, and quotes similar observations of Rosenthal.

Conclusions from consideration of cases of hysterical paroxysmal fever:—

(1) In the course of hysteria, attacks of fever sometimes occur without assignable reason.

(2) These febrile attacks are attended, according to their intensity, by moderate elevation of temperature.

(3) In many cases the fever accompanies a number of severe symptoms which are incidental, and have no common origin with the elevation of temperature.

(4) Attacks of fever may be combined with, but are not dependent upon, convulsive attacks.

(5) "Hysterical pseudo-fever" as well as "hysterical continued fever" may be combined with this form.

(6) This form occurs both in simple hysteria and in hystero-epilepsy.

Ætiology.—Hysterical fever is to be considered as a symptom of this functional neurosis, and any cause which can evoke hysteria can produce hysterical fever.

To sum up—

(1) There is a true hysterical fever which occurs in two forms: (1) continuous; (2) paroxysmal.

(2) Hysterical is to be considered as a functional fever, and a phenomenon analagous to other hysterical symptoms.

(3) It may occur either in simple hysteria or in hystero-epilepsy,

Hysterical Polyuria.—Dr. Albert Mathieu[23] observes that hysterical polyuria is not a very rare affection; it generally affects men. As a rule, the quantity of urine passed is from four or five to seven or eight litres daily, but it may reach eight or ten litres. There is more or less thirst, according to the amount of urine passed. After the polyuria has lasted some time the patients suffer from weakness, partly arising from disturbed sleep, from

anæmia, and loss of flesh. There is polyphagia, but not so marked as in diabetes mellitus. Lately, the author has come across two cases in which larger quantities of urine were passed. A young man of 25, in the Hospital Hôtel Dieu, passed 25-30 litres of urine in twenty-four hours. The quantity was some time after reduced to three litres on M. Thiroloix's giving him some cachets of common salt, having succeeded in persuading him that it was a powerful remedy against polyuria. This cure by suggestion is the more interesting on account of the patient presenting no signs of hysteria.

Another man of 30, in the Hospital Tenon, whom M. Babinski had cured, by suggestion, the year before of paralysis of the radial nerve, was affected with polyuria after a blow on the head in a fight. He passed 20-25 litres of urine daily, and the author was able to reduce the polyuria momentarily, by suggestion during hypnotic sleep. There are, therefore, two types of the disease; in the first, the average quantity of urine per diem is 5-6 litres; in the second it is 20-30 litres.

M. Ehrhardt finds that the urine is pale in colour in these cases and contains no albumen or sugar. The amount of phosphoric acid is normal; that of chloride of sodium is increased in proportion to the quantity of urine passed. The amount of urea is normal or increased, according as polyphagia is present or not. As polyphagia is generally present, the quantity of urea is generally increased.

The reasons which lead to the conclusion that a given case of polyuria is hysterical in nature are: (1) its coincidence with other hysterical phenomena; (2) the mode of onset; (3) its disappearance and re-appearance under the influence of suggestion.

Under (2) polyuria may result from a strong emotion, a great disappointment, and violent injuries, especially to the head. M. Ehrhardt saw it occur twice after the ingestion of iced water during sweating; and after copious libations. It is certain that many cases of diabetes insipidus occurring after physical or mental shock are really dependent on hysteria; the causes enumerated above are the common ones of diabetes insipidus, and together with the experiment of Cl. Bernard, who produced polyuria by lesions of the fourth ventricle, show the neuropathic origin of the disease.

From the point of treatment, it is important to recognise the influence of suggestion in various forms. Sedatives such as opium, belladonna, valerian and bromide have had good effects. We have seen that small doses of chloride of sodium brought

about a remarkable cure. But whatever drug is ordered, the physician must have the air of the greatest certainty as to its efficacy. Valerian and bromide should be given in large doses, accompanied by another element in massive doses, that is, suggestion.

Dr. Tenien[24] gives a full account of a number of interesting cases of hysteria. His views on hypnosis are that it is useful only in hysteria, that cases cured by it are hysterical, and that if a case of apparently organic disease is relieved, this is due to the coincidence of hysterical symptoms.

He attributes the frequency of hysteria in La Vendèe, relatively to other country districts very great, in part to the frequent consanguineous marriages.

Mixed Forms of Hysteria and Neurasthenia (Löwenfeld).—(1) Cases of hysterical or hystero-neurasthenia. Typical neurasthenic condition, generally of cerebral or cerebral-spinal form, together with single and generally slight hysterical symptoms. A special form is an hereditary one in which different anxious states occur, *e.g.*, agoraphobia and temporary spasms of the pharynx of such obstinacy as to render the taking of nourishment difficult.

(2) Cases in which signs of neurasthenia first appear, and then those of hysteria show themselves and proceed to greater relative development; the latter may be ushered in by a hysterical attack. In many cases a hypochondriacal or melancholic temperament is present. This condition has been considered of especial importance in the traumatic neuroses, but may occur in cases of non-traumatic origin.

(3) In which hysterical symptoms predominate, but for a longer or shorter time retire into the background and the signs of neurasthenia predominate.

[Dr. Shaw and Dr. Duryea ("Œdème bleu des hystériques"[24a]) report the case of a young woman who received a blow on her left breast whilst being rescued from drowning, and a few days after began to have convulsions. The seizures averaged two a week for a time, during which the left breast increased in size and became so painful that an incision was made into it, but no pus found. In April, 1892, the breast was enlarged, somewhat pendulous, soft, œdematous, rather cyanotic and intensely hyperæsthetic. There was blue œdema of the left hand and forearm, with coldness. The hyperæsthesia extended from below the breast to the shoulder, and a similar area existed over the left scapular region. There were also considerable areas of anæsthesia on the left side, limited to the anterior aspect of the extremities.

She had numerous convulsive attacks and other hysterical manifestations. The authors remark that œdema, as a symptom of hysteria, although it has been mentioned by a number of authors from Sydenham downwards, very rarely comes under the observation of the profession at large.]

III. Papers on Hysterical and some other Functional Disorders of Motion.

Hysterical Paraplegia.—Dr. Löwenfeld, on the diagnosis of hysterical paraplegia, which often presents considerable difficulty, says that the first thing to ascertain is the presence of actual paralysis. The condition may be one like that of astasia-abasia, or like that found in neurasthenia, in which the patients are afraid to walk because of the pains or disagreeable sensations caused thereby. Further examination may show that loss of power is not present to the extent expected from the patient's statements and behaviour, and with some insistence on the physician's part they may be able to stand and even to walk when they believed that they could not do so.

If paralysis is actually present, the frequent combination of hysterical with organic nervous disease, especially in spinal cord affections, must be borne in mind. The history of the case is important, and it must be remembered that a sudden onset often occurs in myelitis. Pains and paræsthesiæ are common in organic, rare in hysterical paraplegia. Anæsthesia of decided hysterical character may be found. The condition of the knee-jerks, which are very seldom absent in hysteria, is of more importance than that of the plantar reflexes. Escape of the bladder and rectal functions in the presence of severe paraplegia, is against organic disease.

With regard to the supervention of organic charges on long-lasting hysterical contracture, the author remarks that in Charcot's celebrated case in which sclerosis of both pyramidal tracts was found at the autopsy of a patient who had suffered for many years from hysterical spasmodic paraplegia, that this may have been an instance of the frequent combination of hysterical with organic affection of the spinal cord.

Hysterical paralysis of pyschical origin may be due :—

(1) To the non-representation in the cortex of movements of affected parts, so that the movements cannot be volitionally executed, not from a loss of the representations of the movements concerned, but from inhibition of them. Strümpell holds that the

transmission of the wish for a particular action to the central somatic motor (cortical motor) centres is interfered with.

(2) To suggestion, which, as Charcot has shown, may be (*a*) hypnotic; (*b*) traumatic; and, according to the author, may originate (*c*) through the intermediary of sensations of numbness, heaviness, or deadness in the limbs, which are set up in nervous persons by emotional (pyschical) disturbances, and thus bring about the idea of loss of power.

(3) To auto-suggestion (Möbius and others). Such an idiogenetic origin of hysterical paralysis cannot be gainsaid in certain cases, but is not true for all.

(4) The origin of paralysis as a sequel to convulsions is not always due to exhaustion of cortical motor centres, for a fresh convulsion may remove the paralysis, but may be due to inhibition of them.

(5) Lastly, as atrophic paralysis (chronic anterior poliomyelitis) may follow muscular over-exertion, it is reasonable to suppose that in hysterically-disposed persons a condition of functional exhaustion may be brought about in a similar way.

The kind of paralysis which results from special disturbance, or dissociation of the different acts which are accomplished by one group of muscles, whether these movements are simple or reflex, is typically exemplified in the affection which Charcot and his pupils have called astasia-abasia, but as this affection is only one of many, some general term for this form of paralysis is needed. M. Babinski,[25] thinks the term "systematic paralysis of the pyscho-motor functions" or more briefly, as these paralyses are generally, if not always, hysterical, "systematic hysterical paralysis," using systematic in the sense of belonging to a system, would be a suitable one. We fear, however, that this term will not meet with favour, as it seems to us to lack precision, and to be liable to cause confusion, as the term "system" diseases of the cord is well known in quite another (anatomo-pathological) sense.

M. Babinski's first observation is that of a woman of 50, who contrary to what is observed in astasia-abasia, could walk and stand upright, but could not voluntarily perform simple movements of flexion or extension of the toes, feet, or legs, when lying down.

M. Ballet[26] lately published a case in which the patient was affected with hysterical facial paralysis which was only shown in the movements necessary for speaking. M. Parinaud has shown in hysterical ophthalmoplegia that voluntary movements of the

ocular muscles are abolished, whilst the reflex or unconscious are preserved.

M. Babinski reports a case in which when the patient spoke or whistled both sides of the face acted well and equally; the left side of the face also acted well alone, but movements of the right upper lip and right side of the labial commissure were almost impossible. He does not give the case in detail, but we suppose that a limited nuclear lesion was excluded. Another patient suffered, after slight loss of consciousness, from paralysis of the right upper extremity, together with partial loss of sensibility and affection of special senses on the right side. The right side of the buccal commissure lay lower than the left, and the right naso labial furrow was partly obliterated. In any movement of the face, however, the muscles of the right side of the mouth acted well, those of the left side very feebly; this applied both to bi- and unilateral movements. The tongue was put out to the right, but could be moved freely to the left or in any other direction, so that the symptoms were paradoxical. In a third case of a man affected with left hemiplegia, hemianæsthesia and special sense affection, unilateral movements of the face were almost completely abolished, but in the act of whistling and blowing, the muscles of the left side, without executing the movements appropriate to the action, entered actively into contraction, behaving quite differently from a case of organic disease. [This case does not seem to us to be absolutely certainly one of hysteria; the paralysis came on after loss of consciousness lasting six hours, and the patient was a man of 56 years of age.]

The author thinks that in cases of hysterial facial paralysis there is almost always some singularity, foreign to the symptoms in cases of organic disease, which indicates the hysterical nature of the affection, and that the deviation of the mouth is probably due to a contraction of the muscles on the side on which the commissure is highest, and not to a paralysis of the muscles on the apparently paralysed side; that they are cases of spasm, in fact, and not of true paralysis.

Dr. Knapp[27] reports a case of paralysis agitans without tremor, showing the phenomena of propulsion and retropulsion, associated with attacks of inability to walk and with the preservation of muscular force and co-ordination with ability during the attacks to make various co-ordinated movements akin to walking. The author points out that there is not sufficient evidence for the French view that astasia-abasia is a symptom of hysteria. In the course of education, functions such as walking have been assigned to cells at a lower level than those of the cortex, below

the level of consciousness. The process of walking is differentiated from other movements of the legs, as writing from those of the hands. In pure cases of astasia-abasia the motor apparatus is at least to a certain extent in working order. Hammond suggests in these cases loss of power of muscular adjustment, but there is no evidence of disorder of the muscular sense. It is only or chiefly when the action of the highest centres is taken off and the subconscious centres are called into play that abasia becomes manifest. Charcot regards these centres as spinal; Salemi-Pace and Hughes further attribute the trouble to failure of memory in the spinal cells, a partial spinal amnesia. The author finds difficulty in admitting the spinal cord origin of the symptoms, and thinks that the affected mechanism is higher up, and that "we can more easily imagine in the brain, below the highest layer of motor cells or those that act only by conscious volition, a subordinate group of cells in the layer of the subconscious . . . . which has been educated to preside over the movements of walking." A breaking up of the associations between the cells would result in a retention of voluntary movement in the legs, but a loss of the automatic power of walking. He thinks that the results of hypnotism show that "in the field of the unconscious ideas of inability to walk have developed, and by a process of suggestion have acted on the lower cerebral centres for automatic walking." This would bring astasia-abasia under the heading of the association neuroses.

The process is distinct from that of agoraphobia because in the latter some external factor presents itself to consciousness, the attention to it is morbidly increased, and the idea thus aroused dominates the field of consciousness and inhibits voluntary effort. In true insanity of doubt, which is again different from agoraphobia and astasia-abasia, the imperative representation is ever present in consciousness, dominates the entire life and is independent of the patient's surroundings.

In a certain small percentage of cases of astasia-abasia the affection is not an association neurosis, but allied to the intention psychoses—agoraphobia, claustrophobia, &c.

The commonest form is the paralytic; in others the motor disturbance takes the form of spasm or ataxia.

The prognosis is good, especially in children; adults often relapse. The author classifies the varieties in two ways:—

Astasia-abasia (1) { Paroxysmal / Continuous } (2) { With distress (intention psychosis.) / Without distress (association neurosis.) }

So far as the writer's experience of astasia-abasia goes, he has found the common stigmata of hysteria present in adults absent

in children. As regards the nature of the affection, the brain probably acts on the muscles through groups of cells in the cord arranged to form a mechanism for each distinct movement such as walking, the cells discharging in definite order, sequence and time, and so regulating the force, duration and onset of the contraction of every muscle or part of a muscle concerned in the various parts of the movement. Disorder of these cord mechanisms would exactly produce the symptoms of astasia-abasia. We have direct evidence of these centres in the cord, but the existence of "subconscious cerebral centres," distal to or on the efferent side of the cortical motor centres, which the author supposes, is entirely hypothetical.

Möbius,[28] in criticising Blocq's observations on astasia-abasia, observes that he would have done better to have prefixed the adjective hysterical to the name, thus indicating the pathogeny of the malady, and that it is a symptom and not a disease. He points out that astasia-abasia is practically not met with apart from hysteria. He emphasises the fact that the touchstone for all hysterical symptoms is that the patient is unaware of the psychical origin of his troubles. As regards the patient's consciousness, an hysterical is exactly like an organic paralysis; he knows as little of the origin of the one as of the other, and does not distinguish between them. The patient sees that his legs do not bear him, but has no further notion why that is so. This is the reason why hysteria is assigned to neuropathology and not to the psychoses (psychiatry). Scientific researches first disclosed the connection of the hysterical symptoms with (antecedent) psychical processes.

After criticism of Binswanger, Ladame and Blocq's observations, Möbius goes on to say:—

In hysterical loss of power, the idea of the loss of power is the cause of the paralysis. Whether this idea starts up suddenly (as in an accident), or is gradually developed (long confinement to bed), or arises during a dream or in the waking state is of no moment. Its operation takes place on the basis of the hysterical disposition in the one case, and in the other on a special mental state existing at the moment. Of this disposition of mind we have no clear idea. It must be similar to the hypnotic state, and there must be a certain emptiness of consciousness, in which, so to speak, the throne is for the first comer. We know that, besides hypnosis, states of terror and depressing influences (insomnia, hunger) can produce such a condition. If this be granted, we have yet no conception of the manner in which the idea, whatever diseased condition it produces, exactly works, but have to content

ourselves with analogies, which we understand as little, to the paralysing influence of terror and the like. But the clinical touch-stone of hysterical paralysis is that the antecedent causal psychical processes do not enter into consciousness, the patient stands as much outside the paralysis as he does outside one of organic origin, and even the physician, if he is not instructed in its forms and tendency to disappear under psychical influences, may mistake hysterical paralysis for organic disease. Further, it is characteristic for hysterical disorders to affect the young, not seldom children, and to be associated with the so-called hysterical stigmata which indeed cannot be directly caused by idea, but which may be considered to be dependent upon idea in the same way that in the healthy bodily disturbances are dependent upon the affections (emotions).

In their remarks on the first of two cases of male hysteria, Drs. Bourneville and P. Sollier[29] note (1) alcoholism in the father and grandfather and various nervous disorders in the mother, brothers, and sisters; (2) development of hystero-epilepsy in a patient who was late in learning to walk and talk, suffered from nocturnal incontinence of urine, mental irritability and instability, tendency to violent actions, &c.; (3) the patient was a Jew. "During its whole existence, up to the time of the French Revolution, and even now in some countries, this race has for centuries been especially exposed to a number of strong emotional excitants, which have endowed not only individuals but the race itself with an ethnical nervous state, rendering its members particularly predisposed to all nervous maladies." In support of this opinion there, the following local statistics for the Department of the Seine are adduced. In 1875, at the Bicêtre Asylum there were fourteen Jews out of 538 insane patients. If the proportion at the other asylums was the same, that would give 188 Jews out of 7,248 patients. The total population of the Department is 2,220,060 persons, of whom 24,319 were Jews. The proportion of insane was therefore, in the case of Jews, seven per 1,000, whilst for the rest of the population it was only 3·2 per 1,000. (4) When the convulsive attacks came in succession during a rather long period, the temperature rose a little above 38°, but never attained the height it reaches in the status epilepticus.

Professor Popoff[30] records the case of a patient, a peasant of twenty-one, suffering from regular, rhythmical tremor of small amplitude, increased by observation, by attempts to walk or to make any voluntary movement. The tremor could not be voluntarily controlled, and was increased markedly if efforts by the attendants were made to stop it. There was loss of

power and sensation in the limbs, and the fields of vision were contracted. Mechanical irritability of the muscles and the tendon reflexes were increased. The unusual feature of the case was that since the age of eight the patient had been liable to attacks of trembling in the legs, with loss of sensation over them. For some days after the attacks, a feeling of fatigue prevented the patient from working. The attacks came on at intervals of one month, owned no apparent cause, but could be provoked by emotion, *e.g.*, a fright, and lasted two or three days. They ended quite suddenly.

Professor Popoff satisfied himself from personal observation, that the attacks had precisely the above characters, and points out the singularity of their periodicity and of the absence of any exciting cause for them; he can find no exactly similar case on record, but one reported by Chambard (*Hemichorée et tremblement hystérique. L'Encephale*, 1881), most nearly resembles it, as tremor was the most prominent symptom of hysteria present, and came on in attacks, but these had no regular periodicity, and were mixed with hysterical seizures of another kind.

Dr. Kinnosuke Mirna[81] has collected twenty-eight cases of flaccid hysterical monoplegia from medical literature between the years 1885 and 1890, to which he adds three cases of his own. Twenty-three were men and eight women; for the most part the patients were engaged in laborious occupations, involving much muscular effort; six of them were exposed to the risk of lead poisoning. In thirteen nervous inheritance was clear, in eight doubtful, in nine the family history was unknown. During childhood the majority had suffered from incontinence of urine, moral perversions, convulsions, &c. Three patients were alcoholic, there was a relapse of the paralysis in four, and in several hysterical convulsions.

An injury was the most common exciting cause. In six cases there was plumbism; in three a moral emotion preceded the development of the paralysis; two patients were attacked after sleeping in cold and damp; and in two the paralysis appeared after fixation of the limb in a fracture apparatus.

Paralysis came on either immediately after the injury, or after an interval of time, varying from one to twelve days.

Affections of Sensation.—Hemianæsthesia in nine cases; in fifteen the anæthesia was limited at the root of the limb affected by a line drawn perpendicularly to its long axis, but in four others (brachial monoplegias) it affected also the same side of the head and neck and the upper part of the body as far downwards as the epigastrium or umbilicus. In one case (of the author's) there was no anæsthesia present.

Trophic disorders were absent in eighteen, present in nine, consisting of atrophy of the paralysed muscles, and in two also of the bones; in one there was an elevation, in two lowering of the temperature of the paralysed part.

Tendon reflexes were exalted in nine, diminished or abolished in nine, normal in three, and in fourteen cases not recorded. Special senses: in nineteen cases contraction of the visual field, in seven monocular polyopia, in four diminution of acuteness of vision, in two dyschromatopsia; enfeeblement or loss of taste in eighteen, of smell in seventeen, of hearing in twelve. In two patients special senses unaffected; in three not recorded.

Other signs of hysteria noted are, ten times anæsthesia of the pharynx, nine times tender or hysterogenic spots, seven hysterical attacks, and four times "ovarian" tenderness. In twenty-two cases the condition yielded to various methods of treatment; in four it was very obstinate, persisting in spite of all efforts, and in five there were relapses.

Under the title of *Akinesia algera* Möbius[52] describes an affection which consists of loss of voluntary movement on account of pain on movement, without there being any tangible ground for the pains. It appears in people of neuropathic tendencies, family and personal. After over-excitement, a condition of nervous weakness develops. Pain *first* affects only the larger, afterwards nearly all movements. In part it accompanies, in part follows, movements and not only in the moved, but also in other parts of the body. At last there comes complete loss of power, in which the parts appear paralysed and helpless. In addition, the signs of neurasthenia appear: disturbed sleep, weak voice, incapacity for mental exertion, pains and pressure in the head, painful sensations in the back. Undoubted hysterical symptoms were not present in the patients observed. As to the issue that is not as yet determined. We must admit that recovery is possible, yet the history of the two patients proves that mental disease may be added to akinesia algera. The author hesitates to draw general conclusions from only two cases.

I.—The first patient, age 33, was a school teacher. The mother and her family were healthy. The father suffered from paranoia, hallucinations and delusions of persecution, and lost his employment. The family life was much prejudiced by want and the (mad) humours of the head of the house. The patient had thus a melancholy bringing up, and was often terrified by his father. He did very well both at school and university, worked immoderately, and gave great promise for his future career. On becoming a teacher he overworked himself, and was esteemed

by the authorities and beloved by his pupils as an ardent, careful teacher. Soon after obtaining his post he married. In 1887 he got a pain in his head and suffered from sleeplessness. After a holiday he was, for a short time, better. In 1888 the head pressure returned, and mental work became impossible. He could still conduct a conversation and write a letter, but all heavy work and original or consecutive thought was impossible. "The head simply forbade it." In spite of several trials of different methods of treatment his condition grew worse; after coming out of an institution for nervous disorders he passed for three weeks into a peculiar condition of which he had no recollection afterwards, and in which he took no notice of his surroundings, except occasionally to bewail his fate. Treatment by prolonged warm baths somewhat relieved his symptoms. In the spring of 1889 he was ordered to take short walks in the garden, and treated by overfeeding and rest; this resulted in improvement, but he had to break off the treatment and return to a university institution. Here he was ordered to carry out movements with his limbs, and this made his condition worse, he suffered pains in the back, "muscle pains," and "nerve pains." His arms were now affected, and small movements of the hands gave rise to long-lasting pain. A fresh course of treatment by rest gave no better result, and in September, 1889, the author first saw him.

The patient lay on his back. The legs could be moved in every direction, but the movements were feeble and of small extent; no resistance to passive movements. The arms and fingers were in position of extension; movements of both the patient only accomplished to a small extent. Passive movements of the hand and fingers met with a dead resistance, but the patient would not permit much display of force. The patient refused to kneel or to get up. Movements of the head were free and power good; he moved his head without hesitation, and did not complain of fatigue of the neck muscles.

The nutrition of the muscles was in general not bad, but there was slight though evident atrophy of the interossei in the left hand. Skin reflexes normal. Tendon reflexes normal in the arms, very brisk in the legs. Ankle clonus distinct in the left, faint on the right side. No diminution of sensibility anywhere, on the hands and forearms was some hyperæsthesia, light touches being felt as painful. The skin was normal except over the fingers, where was a high degree of "glossy skin." There were no disturbances of the special senses of any kind. The mental condition was unaffected; the intellect clear and good; he could only read a short time, or

dictate a short letter; if he tried to do more pains in the head came on and rendered all thought impossible. K. felt that he must avoid all movement on account of the pain excited. The pains were directly related to the amount of movement, were worse in the arms, and, at the same time, he was not quite free from pain when completely at rest. Some days he was much worse than others. Appetite and sleep were good; the bowels acted regularly, and sexual desire was not entirely lost. Lungs and heart normal.

Passive movements systematically carried out made him so much worse that they had to be desisted from. Various drugs were tried, including two thorough trials with opium, without any effect on the condition. Attempts at hypnosis failed. After some months there was some improvement: the pains in the head disappeared, and he was able to read for long periods. He regained some power of movement in the legs, the tendon reflexes were less exaggerated, but the hands did not improve. In 1890 he had an attack of influenza, which did not affect his general condition. After this he showed very gradual improvement, until at last he was able to move himself from his bed on to a sofa placed alongside it. During the author's absence on a holiday, his *locum tenens* said that possibly K. was a simulator. This deeply grieved him, and he went back, but again very slowly made some degree of recovery. In January, 1891, the condition is that he lies on his back, can move the legs freely in bed but cannot stand a walk; he does not use the hands, but can carry out small movements of the fingers without pain; he has to be fed and uses the bed pan; no muscular wasting nor increase of tendon reflexes, the mental condition is normal, except for a certain irritability and readiness of fatigue.

II.—Miss L., a music teacher, 43 years old. Her mother's family was nervous and the mother herself was paralysed during the last years of her life. Up to 20 years of age L. was healthy, but eccentric. She had attacks of "trembling spasms" after the death of her parents. She was studying music very hard in her 20th year when the pains in her hands began, and grew worse, so that she could not use them; there was also contraction of the hands, so that she had to keep them in position of flexion. She was incapable of any mental exertion, and slept badly. A year afterwards pains in the legs began. At times the pains were worse, so that the joints could not be moved. The illness lasted ten years. Dr. Bärwinkel, under whose care she was, considered the symptoms to be due to general hyperæsthesia of the joints, accompanied by contractures of the fingers, hyperæsthesia of the lower extremities and vertebral joints; hysterical

attacks, lasting some seconds, with loss of consciousness, and clonic spasms of the upper extremities, occurred. Treatment had no influence. She recovered, and for ten years remained well and earned her living by teaching the pianoforte. In the summer, 1889, she strained herself by carrying a handbag on a journey, and also in nursing a sick niece, to which she attributed the fresh attack, which came on with pains in the hands and back, and excitability and weariness. By the middle of October she was already bed-ridden; Dr. Möbius first saw her in November, 1889. The fingers and hands were kept in semi-flexion; as soon as the flexion at the wrist-joints was gently overcome they opened themselves automatically. Passive extension met with no noteworthy opposition. Movements of the hands and arms could be carried out, but with pain, which lasted for some time afterwards in hands and elbow-joints. The legs were freely movable. She could stand and walk from one room into another. This was the extent of her powers, any further efforts giving her pain. There was no disturbance of the special senses, of common sensibility, or of the superficial or deep reflexes. No muscular wasting. Appetite good. Bowels regular. Sleep bad. Catamenia regular. The mental state showed no signs of disturbance. The patient's friends described her as an intelligent, somewhat cold, very strong-willed person. In January, 1890, she had the influenza, which almost seemed to have a good influence over her condition; but some weeks later a bad attack of migraine left her worse than before. Attempts at hypnosis completely failed. Then came a period of improvement, in which the patient was able to stand a little, to sit up, and to read. She went into the hospital, and there passive movements were carried out which resulted, for a time, in improvement; but she relapsed after a short time, and attributed this relapse to being obliged to carry out movements in spite of the pain caused thereby. She made two attempts at suicide, lay motionless on account of the pains that arose from every movement, could no longer sleep, and had lost all hope. Subsequently she became demented, heard voices, had delusions, and thought her food was poisoned.

The two cases correspond very closely in the chief points. Both patients had a bad inheritance; both were, before the illness, already "dégénérès," and suffered from mental over-excitement. The symptoms came on gradually, together with those of neurasthenia. In K. the latter were well-marked, and in L. there were also hysterical symptoms. A slight difference between the two lies in the fact that L.'s first illness began with a contraction of the hands, and not with loss of voluntary movement; but of the second illness, which alone was observed by the author himself,

there was no sign of contraction, the symptoms being similar to K.'s. Further, in K.'s case there was exaggeration of the deep reflexes and wasting of the muscles of the left hand. The last might be attributed to the compression of the bandage, or be dependent on the disease. The latter appears to me to be the more probable, and both symptoms might be due to hysteria, for a like degree of excessive myotatic irritability and muscular wasting has been shown by Charcot and his pupils to occur in hysteria. In both patients the pains were very obstinate, and all attempts at compulsion and the influence of emotions made them worse. The first case has not yet ended, and insanity has brought the second to a provisional ending. The question first of all arises, what is the nature of the morbid process? The inherited neuropathic taint, the presence of nervous hypererethism, of neurasthenic and hysterical symptoms, the peculiar course and the general features of the disease lead to the conclusion that it is a psychosis. If we attribute akinesia algera to paranoia in the widest sense of the word, a further definition of the variety is also desirable. The conception of neurasthenia would have to be extended to include akinesia algera; and one can, on the other hand, adduce grounds for the hysterical as well as for the hypochondriacal nature of the symptoms. All depends on what we mean by these terms. Briefly one may define as hypochondriacal, those bodily disturbances of whose psychical mechanism the patient is conscious; and as hysterical, those in which the connection between perception and material disturbance does not enter into consciousness. In this sense the pains of akinesia algera are hysterical. Should exception be taken to consideration of the pains as material symptoms, the reply is that deficient sensation and hyperæsthesia, though really subjective, are commonly reckoned as material symptoms. It might be further objected that the estimation of a symptom as hysterical does not depend on theoretical speculations, but on the presence of other undoubted signs of hysteria. This view, however, is contradicted by the existence of many forms of monosymptomatic hysteria. Often the diagnosis of hysteria can only be made by exclusion.

Although Möbius regards the pains of akinesia algera as hysterical, he cannot consider the disease as simple hysteria. There is an analogy between cases of analgesia algera and those of general anæsthesia, which have lately been published by Kinkenberg, Heyne, and von Ziemssen. Hysterical manifestations in analgesia algera are combined with hypochondriacal and neurasthenic symptoms, and taken as a whole the disease resembles more closely severe hypochondriasis than simple hysteria. W. Neftel[35]

is quoted by the author as having described a condition very like that of analgesia algera under the name of atremia. In these cases (four in number) the patient had always a nervous heritage, and fell ill through over-excitement; there were photophobia, incapacity for mental or corporeal exertion, because both evoked general indisposition, paræsthesia, diarrhœa, and loss of sleep. The patients lay continually in bed, and all attempts to make them walk aggravated the symptoms. Treatment was without effect. Neftel considered atremia as hypochondriacal insanity. The chief difference between atremia and analgesia algera is that in the former, walking, standing, and sitting are impossible, whilst the limbs can be freely moved in bed without complaint, whilst in the latter all movements of the limbs are entirely desisted from. Atremia bears the same relation to analgesia algera as abasia-astasia to hysterical paraplegia. In atremia walking produces only disturbances in the general condition, and paræsthesia in the head and back, whilst in analgesia algera the pains are in the parts moved. In other respects the two conditions correspond. Binswanger describes a similar condition of loss of voluntary movement on account of pain, and regards it as an episode in the the course of hysteria. As already mentioned, so far as the above observations go, treatment has proved ineffectual and the prognosis is bad.

Erb[94] thinks that it is still doubtful whether the symptom-complex so clearly painted by Möbius can be regarded as a distinct morbid entity; time and further observations will show, but there is no doubt as to the practical utility for the advance of knowledge in thus circumscribing a group of symptoms. As Möbius points out, the affection on the one hand approaches the psychoses, and on the other is closely related to hysteria, and especially to neurasthenia. As for the cases that Erb can recall to memory, he has referred them partly to the severe forms of neurasthenia, which especially in effeminate persons present the highest grade of irritable weakness and exhaustion of the nervous system, partly to that affection which we call spinal irritation. They either affect undoubted hysterics, or they appear under the form of hypochondriacal psychosis.

He considers that there are essential points of difference between spinal irritation and akinesia algera. Erb has seen several similar cases, and come to the conclusion that it is very difficult to treat them successfully; he reports the following case as illustrating in a striking way the symptoms of akinesia algera.

The patient was an Englishman, aged 47 years, in the diplomatic service in Italy. He came of a highly neuropathic but talented family. The patient was always weak and nervous, and overworked himself at school and college. He had been ill since his twenty-second year; the illness began, after excessive mental overwork, with inability for gymnastic exercises. Later, exercise of any kind caused pains in the heart, palpitation, and various vaso-motor disturbances. In January, 1873, every slight movement brought on pains in the heart and palpitation; he was treated with injections of strychnine, which had an untoward effect, so that he could neither stand nor sit, but took to bed. After fourteen days he was able to stand for a short time, and to sit up longer, and passed six months partly lying, partly sitting. The head was free from pain, and mental work could be well carried out. Attempts to rouse him and to make him exert himself only ended in aggravation of his condition. In the year 1873 he lost the power of reading and of writing, on account of the pains they excited; later he could not even bear reading aloud, though he could still sustain a conversation. He was obliged to keep constantly lying down.

From 1878 to 1888, the patient remained in the same state: he lay in a horizontal position day and night. All the functions of the body were normally performed, except that sleep was bad, and sexual power abolished. From 1888 the patient made again attempts to leave the horizontal position, but only succeeded in standing for the space of four minutes; he could not bear anyone to talk to him, nor for more than two or three words to be said to him, although he could talk freely enough himself. Two new attempts to get him out of the horizontal position failed; as soon as he was raised a few inches the pains came on again. Although he had thus passed fourteen years on his back, he had not lost his sense of humour, and had even made himself some reputation as an author. The patient struck Professor Erb as a highly gifted, intellectual, and amiable man. He could not bear Professor Erb to speak any sentence longer than two or three words, but did not seem to remark that he could quite well bear his own long-flowing speeches. There were no special nervous or hysterical symptoms. There were no signs of organic nervous disease. All forms of treatment had been tried, without any success.

The above case speaks for itself. In the leading features, in the strong neuropathic inheritance it falls under Möbius' definition, and it may be emphasized that in this case symptoms of neurasthenia, hysteria or of any psychosis were absent. Whether the patient from psychical influence or gradual disappearance of his notable hyperæsthesia will ever recover from his lamentable condition, remains undecided.

(*To be continued.*)